THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—22ND YEAR. SYDNEY, SATURDAY, DECEMBER 28, 1935. No. 26.

Table of Contents

SOME ALLERGIC DISORDERS.[1]

By D. L. BARLOW, M.D.,

Honorary Medical Officer to the Vaccine and Asthma Clinic, Adelaide Hospital.

THE object of this paper is to indicate the methods whereby it is possible to bring relief to those who suffer from some allergic disorders. It is therefore my intention to discuss the more important practical aspects of the subject and to refer to the mechanism involved only so far as we are reasonably sure of it, and where it is necessary for a proper understanding of the matter.

A great deal has been written on the subject during recent years, and I am sure that much of what I have to say is already well known to most of you. However, until quite recently there has been considerable divergence of opinion in regard to the importance of allergy in a number of disorders, and many eminent physicians have been sceptical on the subject. Now, however, the main facts are established beyond doubt, and the results of treatment over a reasonably long time enable us to form some definite conclusions.

There has been some confusion over the terms hypersensitiveness, allergy, anaphylaxis and the rest, and authorities are not yet all agreed on the limitations of these terms. The terms protein sensitivity and hypersensitiveness may be used to refer to any condition in which an increased or abnormal response occurs on contact with a foreign protein; allergy is generally understood to refer to conditions of hypersensitiveness occurring naturally, whereas anaphylaxis is a convenient general name to include all those similar manifestations which can be brought about artificially, as a rule for experimental purposes. Although the symptoms appear to be due to the same type of reaction in both, there are certain differences between allergic and anaphylactic phenomena which are worthy of attention.

[1] Read at a meeting of the South Australian Branch of the British Medical Association on August 29, 1935.

In the first place, the symptoms resulting from allergic reaction may be very prolonged and comparatively mild, but anaphylactic reactions are acute and run their course either to death or recovery in a brief space of time. This difference depends to some extent on the mode of contact with the offending protein; for instance, there is an enormous difference between inhaling a few grains of pollen and having an extract injected into the blood stream. Cases have been reported in which asthmatics, receiving an injection of serum, such as antitetanic serum, have died rapidly in a condition corresponding closely to that of experimental anaphylactic shock in animals. Another feature of allergy, but not of anaphylaxis, is that typical symptoms sometimes arise from contact with non-protein agents, for example, aspirin. The explanation appears to be that a compound is formed by such a chemical entity and some tissue protein, and that this compound acts as an antigen in the same way that it has been proved that attached carbohydrate groups determine the types of pneumococci.

It appears to be partly in this way that asthma and dermatitis are caused in some men who work with timbers, such as red pine and blackwood. In an investigation a few years ago I could not find any evidence of sensitization to specific protein of the wood, and other people have had similar experience.

We now come to the most difficult problem of accounting for the fact that, although everyone is constantly coming in contact with foreign proteins of all sorts, yet only a small proportion of the community displays symptoms of allergy. About all that is known is that there is usually an inherited tendency and that the constitutional difference from the normal is the general liability to become allergic. The reaction may be manifest as asthma in the parent and as hay fever in the child, or *vice versa*, and the offending proteins may be of quite different groups. The frequency and degree of contact have much to do with determining to what proteins the patient becomes sensitive.

Some persons not infrequently lose their sensitiveness to a particular protein apart from treatment, especially if they are no longer coming into contact with it. They may at the same time gradually acquire sensitiveness to other proteins. In general the tendency to acquire sensitiveness is greatest in childhood and gradually decreases. In infancy sensitivity to a protein may develop as a result of minute quantities of unsplit foreign proteins being carried over into the mother's milk, and in this way symptoms may arise very early in life.

Mechanism of Allergy and Anaphylaxis.

The symptoms are due to an interaction of antigen (that is, the sensitizing agent) with its corresponding antibody in contact with a tissue cell. This sort of interaction more usually occurs in the tissue fluids or blood plasma and then causes no disturbance; but for some unknown reason, in a certain proportion of people antibodies tend to remain attached to tissue cells, and these people are liable to symptoms when they come into contact with the offending protein.

It is now accepted that the interaction of antigen and antibody results in liberation of histamine. It has been shown that some tissue cells contain an appreciable quantity of this agent.

Histamine has well recognized toxic effects. It causes contraction of unstriated muscle and causes swelling and œdema of other cells. The nature of the symptoms in a given case depends partly on the location of the cells which are most sensitive. This location varies in different persons, so that a protein absorbed by the alimentary canal may in one person cause eczema and in another asthma.

History of Allergy and Anaphylaxis.

Jenner in 1798 noticed an erythematous reaction in the skin of a person who had suffered from smallpox or cowpox when variolous matter was inoculated. Richet, about the year 1900, recognized that a condition of hypersusceptibility was brought about in animals by inoculation of protein, and he first used the term anaphylaxis.

Von Pirquet in 1906 concluded that an antigen-antibody response was involved in these phenomena. As regards hypersensitiveness naturally acquired, Blackly, of Manchester, in 1873 had obtained definite reactions to grasses on his own skin and mucous membrane, and in 1890 designed a first-rate pollen filter.

Curtis in 1900 attempted desensitization with pollen extract with some success. This method was carried on by Freeman in London and by Cooke in America, and from about 1913 onwards a vast amount of work on desensitization in asthma as well as hay fever was undertaken in the United States of America, and excellent results were reported. That work has since been confirmed in most other countries.

It is interesting to note that, although the method of sensitization by inoculation of specific proteins originated in England, the leaders of the profession there were very slow to recognize its value, even after the Americans, in their characteristic fashion, had taken it up and greatly improved its technical results.

The practical results of application of knowledge concerning desensitization were widely recognized in Australia and South Africa at a time when the Asthma Research Council in England was still in doubt. Its recent reports have in every way confirmed our results.

Other Factors in Allergic Disorders.

In conditions of an allergic nature it is most important to realize that numerous other factors may be concerned in the causation of symptoms. Of these the most important are physical or chemical irritation of the part affected, and factors operating through or involving the nervous system.

In asthma reflex irritation from stimulation of the mucous lining of the nose is sometimes a contributing cause, but its importance has been greatly exaggerated.

The psychological element is often of some importance, and occasionally of very great importance, and it is the most difficult to combat in treatment. Chronic infections in producing asthma may act in several ways. A focus of infection, such as antral disease, may give rise to sensitization to a bacterial antigen or to products of bacterial activity. It may have some influence by reflex irritation affecting the bronchial musculature. Infection of the bronchial tubes may allow foreign protein to gain entry to the tissues more readily; it may give rise to bacterial sensitization, or, by setting up an irritable condition of the tubes, may exaggerate the effect of other factors. It may act in all these ways at once.

Although the amount of bronchitis accompanying the asthma varies greatly in any series of cases, I have not been able to find any justification for the attempt to subdivide asthma into the allergic and the non-allergic or bronchitic types. Of course, chronic bronchitis with emphysema can give rise to considerable dyspnœa, but these cases need not confuse the issue. Asthmatics who cannot be shown to be sensitive to foreign proteins (other than bacterial) nevertheless have an eosinophilia in the blood, just as the admittedly allergic people. In hay fever also, in addition to the allergic basis, we have the factors of nasal abnormalities, infection of the nasal cavities or accessory sinuses, direct irritation by dust *et cetera*, and the phychological element.

It will now be more useful to consider in some detail the more important clinical manifestations of allergy.

Asthma.

Asthma is our biggest problem, and it is in this ailment and in hay fever that the recent work has done so much in yielding results not formerly attainable.

One point in regard to diagnosis seems worth mentioning. I have on occasions seen patients concerning whom confusion as to the diagnosis had arisen because their dyspnœa appeared to be greater on inspiration than on expiration. There is really nothing unusual about this in the milder cases or at the begininng of an attack. When the spasm commences, the patient first notices difficulty in getting air into the lungs, but as the lungs gradually become distended through failure of the elastic recoil in expelling the air, expiration becomes a conscious effort and finally becomes much more laborious than inspiration.

The Causation of Asthma.

There are many factors in the causation of asthma, though not all are involved in any particular case. It will not be possible to consider all the factors in detail.

An allergic basis is the main cause in the majority of asthmatics. Apparently there are still people who doubt this, but as time goes on their number becomes less.

The question of bacterial sensitization is a difficult one and has not yet been fully elucidated, but instances of extreme sensitiveness to bacterial antigens are not infrequently encountered.

In one patient, whose skin gave a very strong immediate reaction to an autogenous *Staphylococcus aureus* vaccine, an original dose of one million organisms caused a definite attack of asthma. The dose was next reduced to fifty thousand, but it was found that this could be increased by about twenty thousand each time for a number of doses without giving rise to slight reaction. The results of treatment were entirely satisfactory.

Products of bacterial activity, rather than the organisms themselves, may at times produce sensitization.

The number of foreign proteins which may cause sensitization is, of course, unlimited, and the number which occasionally do cause asthma is rather large; but in the great majority of cases the condition is caused by some very common protein, such as people are constantly encountering in their food or in the air they breathe, or in other ways.

The nature of the offending proteins in an individual case depends to a large extent on occupation, environment *et cetera*, for these determine the things which may be frequently absorbed into a patient's tissues. There is often a history of prolonged contact with the offending protein before symptoms arose. More often than not several proteins cause a reaction, but there is a definite tendency for these to belong to one group, for instance the animal epidermal group.

In childhood, one of the commoner foods, such as cow's milk, egg or cereal, may be the all-important factor, but in my experience this is not very usual. Even in quite young children epidermal proteins of domestic animals and birds frequently cause asthma. The sensitization no doubt arises as a rule from feathers and hair used in the manufacture of bedding, clothing and toys. In this country horse and cattle dander are the most frequent of the animal offenders, cat and dog dander being also important.

As age increases, the epidermal and pollen proteins together account for a high proportion of cases, and many people are found to react to extracts made from household dust. Some of these patients react to various dander and feather proteins, and it may be the presence of small quantities of these proteins that accounts for the reaction to dust extract. This is not, however, the whole explanation, as a number of people give positive results with the dust only. It is considered by some authorities that in such circumstances minute atmospheric fungi are the real cause.

The method of preparing dust extract from the patient's home or workshop may be of interest. If possible, a quantity of dust is taken from a vacuum cleaner after a general spring cleaning. This is added to a suitable volume of Coca's fluid or other solvent for about ten days. Filtration is then carried out by means of a Seitz pressure filter to obtain a germ-free fluid. Tests for sterility should be made. This is standardized as regards nitrogen content and dilutions are prepared for testing and treatment. Pollen and food extracts are prepared

in a similar way, but there are several alternative extracting fluids; one of these, which contains glycerine, is useful for extracts which are to be kept as stocks for testing, as the proteins appear to be more stable in glycerine than in Coca's fluid. The latter consists of saline solution with the addition of mono-acid and di-acid sodium phosphate to form a buffered mixture, *plus* enough phenol to prevent bacterial growth.

The particular pollens responsible locally for allergic disorders will be discussed under hay fever.

Methods of Testing.

The dermal or scratch test is the most useful all-round test and should always be used with pollen and epidermal proteins before inoculation methods are tried. As a rule, dry pure proteins, specially prepared, are used and liquefied on the skin in a drop of either Coca's fluid or decinormal alkaline solution. The scratches are made about two millimetres long and not quite deep enough to cause bleeding.

Certain materials, such as egg white and milk, may conveniently be tested on a scratch in their natural condition. The use of prepared extracts in the scratch method is not recommended, as, although proteins giving strong reactions will be discovered in this way, many very important reactions will be missed; if this method is used, no reliance must be placed on a negative result. A reaction consists of an erythematous area at the site of application of the protein, anything from a quarter of an inch up to several inches in diameter, with or without a definite wheal in the centre; the most characteristic reactions have irregularly shaped wheals.

The degree of reaction is not necessarily proportionate to the severity of the symptoms, as the skin may be highly sensitive and the mucous membrane only moderately sensitive, or *vice versa*. Reactions usually develop fully within half an hour. The interpretation of the result is in many instances perfectly simple, as the reactions are obvious and easily correlated with the clinical history. In many cases reactions are not so obvious and yet may be most important; and their interpretation is largely a matter of experience.

Intradermal testing is carried out usually when dermal testing gives negative or indefinite results, and concentrations as high as 1 in 200 may be used. With a short bevel fine hypodermic needle sufficient solution is introduced beneath the epidermis to raise a little lump about two millimetres in diameter. A control injection, in which solvent only is used, is always given. Reactions are noted within half an hour and are similar to those obtained by the dermal method, except that the wheal is usually more prominent. The reactions from both methods of testing fade as a rule in an hour or two.

If a strong solution were used without a dermal test first being made, general reactions could be caused, and therefore caution is necessary.

A third means of testing is the patch method, in which a paste of the material (usually food) is incorporated in a small pad of lint or gauze and applied to the skin and fixed on for twenty-four hours under a covering of oiled silk. This is more applicable in urticaria *et cetera* than in asthma.

Apart from deciding that the condition is one of true bronchial asthma, a general examination and the taking of a history to determine various factors which may be operating are essential. Focal infections, such as antral disease, may be helping to keep up a chronic bronchitis, or the patient may be sensitized to bacterial products. The effects of occupation and environment must be carefully studied.

Treatment.

It is not my intention to go into details of general management, drug treatment *et cetera*, but I shall merely mention the necessity for doing everything possible in the way of regulating the patient's activities and improving his general health. The psychological aspect is worthy of special attention, as a reasonably hopeful attitude of mind is certainly distinctly helpful. We do not know how to alter or to influence directly the underlying defect which causes the patient to become sensitive, but in this direction there is a possible hope for the future.

The offending proteins are avoided as far as possible, and here may be mentioned the advisability of care with children of allergic parents and of their avoiding animal pets, feather pillows *et cetera*, which are so liable to cause sensitization. It is impossible completely to avoid pollen and epidermal proteins, which are often carried long distances in the atmosphere. If a patient reacts to any of these, treatment with the object of desensitization is usually indicated. This must be carried out with great attention to dosage to suit the individual patient, for any rule of thumb method is fraught with risk to the highly sensitized patient, and in others may fall short of being effective.

The best way to proceed is to test the skin with proteins in various dilutions, differing by multiples of ten, to discover the strongest dilutions which fail to cause reaction. Inoculation is commenced with 0·1 cubic centimetre of this dilution, and the dose is doubled on each succeeding occasion until 1·0 cubic centimetre of this solution, containing 1,000 units per cubic centimetre, is reached. The unit is taken as $^1/_{1000}$ milligramme of protein; solution *D*, ten times as strong, is then commenced with 0·15 cubic centimetre, followed as a rule by increase of about 50% until, if possible, a dose of 1·0 cubic centimetre is reached. This may then be repeated at intervals of seven to ten days for several months. The exact length of treatment will depend on a number of factors, including, of course, the rapidity of progress of the patient. Stronger solutions than *D* are frequently employed. Freeman, in England, has found that his results have been better since he has used these concentrated extracts. This applies particularly to the pollen-sensitive people, but some of the disappointments which have followed treatment in Australia are without doubt due to the fairly general acceptance of *D* strength

as a maximum for treatment. The interval between inoculations is usually two to three days to commence with, but gradually increases as the larger doses are reached.

Indications for reduced dosage are local swelling and irritation at the site of injection, or temporary increase of symptoms. These must be carefully watched for. If this is done, troublesome reactions are unlikely to occur. Here it must be mentioned that very great care is to be exercised if a fresh batch of solution is used, as, unfortunately, it is not possible to standardize these very accurately, and the new solution may be more potent.

Another practical point is that unusual care is indicated to avoid accidentally injecting the solution into a small vein, for, if this should happen, a very unpleasant reaction might result. A slight pull on the piston of the syringe after the needle has been inserted through the skin will immediately reveal entry into a vein.

Some people make a practice of adding adrenaline to the stronger protein solutions as a precaution against reaction. This certainly allows of more rapid treatment and is valuable in some circumstances, but in my opinion is not to be recommended as a routine.

A question for consideration is how long the effects of desensitization last. It can only be said that patients vary greatly in this respect, but that a great deal depends on the thoroughness of treatment. The difficulty of deciding when to discontinue the inoculations has been alluded to previously; it is, however, desirable to continue until the skin reaction to the strongest solutions has either disappeared or become very slight. Long before this the symptoms may have disappeared, and it is not always easy to make patients see the reason for going on with treatment. In many cases it is advisable to give further inoculations at a later date, although no further symptoms have occurred. This is usually the case with the pollen-sensitive. In order to decide what is indicated, retesting should be carried out from six to twelve months after the initial treatment is completed.

When the patient reacts to numerous proteins there is some difference of opinion as to whether it is necessary to include all of them in the extracts used for treatment. Freeman, for example, is convinced that the only grass pollen which is essential in England is Timothy grass, but I am sure that we cannot simplify matters to such an extent here. When, however, a number of grasses bring about reactions, providing one includes those giving the stronger positive results, there does not appear to be any practical advantage in including the lesser offenders.

A method recently advocated, of combining a number of the common epidermal proteins and of starting treatment without testing beforehand, is mentioned only because of the rather considerable publicity it has had, and in order to indicate that it is not approved by the more experienced authorities.

In spite of adequate treatment by desensitization, the symptoms may not abate as expected. For this there may be several reasons, but, apart from a few very resistant cases, the usual cause of disappointment in these circumstances is the failure to recognize and to deal with some factor other than that of sensitization. Not infrequently a chronic bronchitis is present; this will usually clear up under autogenous vaccine therapy, and not infrequently it is a good plan to combine this with the protein therapy. Most of the patients in whom it is not possible to discover any evidence of an offending protein can be benefited by autogenous streptococcal vaccines made from the sputum or naso-pharyngeal secretions. Most of the organisms are of the non-hæmolytic and viridans types, but it must be added that many of the patients will do well with a mixed streptococcal vaccine made from a number of strains of these types. *Staphylococcus aureus* is, however, sometimes the most important organism, and patients can be extremely sensitive to it. The same applies occasionally to other bacteria.

In the administration of vaccines to asthmatics one must commence with a small dose, usually five million organisms; but if there is evidence of special susceptibility, a much smaller dose is indicated. For this reason it is always better to make an intradermal test with a minute quantity of the bacterial emulsion before completing the preparation of the vaccine. The dose of vaccine given should be increased cautiously, and anything beyond a mild local or focal response is to be avoided. After reaching a moderate number, say fifty to one hundred million organisms, if improvement is steady, it is advisable to go on repeating the dose, but an increase can be made if progress diminishes. Inoculations are made at three or four day intervals at first, but the intervals are gradually increased to seven or eight days.

Other Methods of Treatment.—Several other methods of treatment of asthma succeed at times. These include autohæmotherapy and peptone therapy. I have not found these successful when specific therapy and vaccine therapy have been disappointing, and it is certain that when the latter succeed, many failures would result from protein shock methods.

When for any reason it is impossible to apply the methods advocated in this paper, peptone therapy or hæmotherapy is certainly worth trying. X ray exposures, given according to various plans, are capable of affording at least some temporary relief. even in severe and resistant cases; but there is often exacerbation of symptoms for a time, and I have not found the results to last for more than a few months in these cases.

Acid therapy is a complementary rather than an alternative form of treatment. Its rationale depends on the fact that it has been demonstrated that many asthmatic children and some of the adults have low gastric acid values, and it is thought that associated with this there is an abnormal absorption of undigested proteins.

Desensitization to food proteins is often successful by the method of adding at first minute, then gradually increasing, quantities of the offending food to an acid mixture and giving this before meals. Subsequently it is advisable to see that no lengthy interval occurs without consumption of this food.

Drug Treatment.—In severe attacks of asthma even a large dose of adrenaline solution may fail to give complete relief; in such circumstances it is advisable to continue to give small additional doses (say 0·1 cubic centimetre) at short intervals until breathing is quite free, and then to administer a suitable hypnotic.

Hay Fever or Spasmodic Rhinorrhœa.

There is no need for me to describe the symptoms of hay fever or spasmodic rhinorrhœa. The trouble is usually seasonal and due to pollens of grasses, garden flowers, and occasionally of trees; it may, however, be caused by animal danders, feathers, orris powder (used in the manufacture of face powders) or household dust, and is in these cases more or less perennial. Bacterial infection may be the chief or subsidiary cause of the perennial type.

If the patient is sensitive to pollens, there is usually a quite definite response to a scratch test, affecting an area of 2·5 to 5·0 centimetres (one to two inches) in diameter, but sometimes much larger. In a few cases reactions are elicited only by intradermal injection; this is quite common as regards the dander and feather proteins. Such reactions are nevertheless most important, and just as often lead to successful treatment.

It is uncommon to find a reaction to one pollen only. In this State most sufferers are either sensitive to numerous grasses and cereals, to several members of the Compositæ group, or to members of both groups. The grass-sensitive are rather more numerous than the others. A few people are sensitive to plane tree, wattle, pepper tree, so-called white cedar, and some others.

Although a very definite tendency appears in most hay fever sufferers to become sensitive either to a number of grasses or several members of the Compositæ group, there is considerable difference in any series in the degree of reaction with the various members of these groups, so that in one person the strongest result will occur with, say, couch grass, whereas the next will react strongly with bromus and only to a minor degree with other grasses. There is evidence that both group and individual antigens are present in the pollen grains. Therefore, as Timothy grass produces much more pollen in England than any other grass, Freeman is able to obtain good results by using this alone.

In South Australia the position is different; there is no such predominant grass and the season is much more prolonged, partly owing to the lack of rain to rid the atmosphere of the pollen grains. Thus for many months each year there is a considerable variety of pollen in the atmosphere, and the prevailing type varies from time to time.

For practical purposes, if the extract for treatment is made to include those members of each group which give the larger responses, there is little or no advantage in including the others.

The tree pollens give rise to comparatively little trouble, and most of the people who blame the plane tree are really sensitive to grasses or flowers which pollinate at about the same time as the plane trees. In general terms, the amount of trouble caused by an individual pollen depends on whether it is light and easily carried about by the wind and, secondly, on its degree of prevalence in the particular locality.

Treatment of Hay Fever.

The plan of treatment varies according to whether it is commenced during the pollen season or in between seasons. In this locality, with its prolonged season, it is certainly worth while commencing treatment after symptoms have commenced, as it is possible to minimize them; at the outset very small doses must be used, and the intervals between inoculations should be short—one or two days. As symptoms abate, the amount given can be increased steadily and a fairly high level reached during the season.

To be effective the treatment will need to be continued before the next season, or it may be carried out right through the free season with longer intervals between inoculations. The usual plan is to give pre-seasonal treatment and to work up to as high a dose as possible just prior to the pollinating time. A somewhat smaller dose can then be given at intervals of ten days until the worst of the season is over. In severe cases it is worth the patient's while to keep up treatment, as mentioned previously, throughout the year, until a season has passed without troublesome symptoms. Retesting of the skin occasionally will help to indicate the progress of desensitization; this may be difficult to judge by the patient's account of the severity of symptoms. Details of dosage have been discussed in the treatment of asthma.

Results of Treatment.

Improvement from seasonal treatment may be expected in the majority of cases in the course of a few weeks; with some patients there is considerable and rapid amelioration. Some cases are disappointing.

Pre-seasonal treatment, if adequately carried out, gives complete or almost complete freedom in about 80% of cases, and a varying degree of improvement in most of the others.

Mechanism of Desensitization.

It appears that in the process of desensitization the attached antibodies are gradually removed from the tissue cells, but why this happens is not very clear. In anaphylactic shock these antibodies are rapidly used up, and therefore the occurrence of a second shock is not possible for some time.

The perennial type of rhinorrhœa due to dander, feathers *et cetera*, is treated similarly, but as a rule it is not necessary to commence with such small doses. In the bacterial type, or when infection is thought to be present as a complication,

autogenous vaccines are of considerable value, and here, as with asthma, caution is needed to avoid reactions of undue severity.

Urticaria and Allied Conditions.

There is evidence of an allergic basis in a considerable proportion of cases of urticaria and similar conditions. It is well known that quite a number of people are unable to take certain foods without suffering from an attack of urticaria or angio-neurotic œdema. In many instances the offending article is one of the less common foods, such as oysters, strawberries or lobster, and the association is soon realized by the patient, who learns to avoid the cause.

The more troublesome cases are those resulting from ingestion of the everyday foods, or from what is sometimes termed intrinsic allergy, arising from sensitization to bacterial products or proteins altered by metabolic or chemical means. A moderate number of urticaria patients are sensitive to dander and feather proteins, and one occasionally sees a dramatic result from such simple measures as changing the pillows.

As regards the investigation of these cases, not many patients will be found to react to a dermal scratch test, but a considerable proportion will react to the intradermal method; in some cases, however, the nature of the protein really at the back of the trouble will not be revealed unless we use other means. The patch test is sometimes valuable. It consists of mixing a little of the suspected material with saline solution or Coca's solution, soaking a small square of gauze or lint in this, and applying it to the skin for twenty-four hours, the material being kept moist by being covered with oiled silk. A positive reaction is indicated by an erythematous area.

This test is particularly useful in the case of such non-protein materials as aspirin, and is used also in the investigation of eczema, and less often of asthma and hay fever. It is interesting to note that in this way some of the constituents of ointments prescribed for eczema *et cetera* have been found to give reactions in the patients for whom they have been prepared.

Bacterial sensitization resulting from some focal infection is not an uncommon cause of urticaria, and such foci should be carefully sought, especially if skin testing gives negative results. Following the elimination of these foci, autogenous vaccines may be required to clear the condition.

If the cause is still uncertain after all these investigations, test diets sometimes discover an offending article of food. The test diets consist of a few items which themselves rarely lead to sensitization; and when one of these diets is found on which the patient improves, individual articles are added from time to time. In this way we may come on something which rapidly causes an exacerbation; it can subsequently be avoided. When the causative proteins are discovered, treatment follows the lines already discussed in dealing with asthma.

As to location of infective foci, the teeth, nasal sinuses, tonsils and gall-bladder are the most common so far as present work goes.

There is an interesting point in regard to the inheritance of angioneurotic œdema: there are families in which this complaint has occurred in a number of successive generations, without any special tendency to allergic conditions, such as asthma, appearing in the family. It was indicated previously that in allergy it is the tendency to allergic conditions in general rather than to any particular manifestation which is inherited, and so far no evidence of an allergic basis has been produced in regard to these families. In childhood urticaria, which so often appears to be due to certain articles of diet and which often coincides with digestive derangement, is not in all these cases associated with sensitization, but apparently with various metabolic products *et cetera*, which exert a direct toxic effect.

Here may be mentioned the frequency with which children gradually become less sensitive to food proteins until they usually become spontaneously cured. Many of these children improve when a properly regulated diet is instituted (bacon and other pig products are to be avoided). When general measures fail to give relief, tests for sensitization should be made.

Eczema and Trade Dermatitis.

Eczema is due to sensitization in a high proportion of the infants affected, but in a progressively smaller percentage as age increases. In early infancy food proteins cause most of the trouble. The child may become sensitized by the passage into the milk of minute quantities of unaltered foreign protein, or it can become sensitized *in utero*. Egg white, milk (cow or goat), wheat and oat are the commonest foods involved. In later infancy epidermal proteins are frequently responsible, at least in part. Most of these children are inclined to have a subnormal gastric acid secretion. The child may be able to take boiled milk without ill-effect, as the lact-albumin which causes most of the mischief is thereby coagulated.

The administration of a hydrochloric acid mixture and, if the protein cannot be avoided for any prolonged interval, the addition to this mixture of minute but steadily increasing quantities of the protein will as a rule rapidly improve matters. The tendency toward spontaneous recovery without special treatment is well known, but does not occur as a rule for months or years. If the causative protein in eczema is one of the epidermal group, desensitization by means of inoculations should be undertaken and gives good results.

Migraine.

The tendency of migraine to afflict certain families from generation to generation is well known, but as a rule these families do not suffer from the recognized allergic conditions, such as asthma. Naturally there will sometimes be an allergic family history; this is found in a large

section of the community, and coincidence would thus account for some such histories. It is reasonable to believe that allergic reaction plays a part in the causation of some cases of migraine, but there is no evidence to prove that it is a big factor in any appreciable proportion. Eosinophilia is absent from the blood as a rule, and no large number of patients give positive skin tests. When there is evidence of allergy, however, it is doubly important to treat the allergy.

Epilepsy.

There has been a good deal of speculative writing regarding a supposed allergic basis in epilepsy, but the subject is mentioned here only to indicate that nothing in the nature of proof has been adduced and that no advance in treatment has been made on these lines.

Conclusion.

In conclusion, I should like to emphasize the following important practical considerations, which have been referred to as having a large bearing on the results achieved in the treatment of allergic disorders:

1. Testing for specific offending proteins must be thorough, not so much in the employment of an extremely extensive range, including rare proteins with which the patient scarcely ever comes in contact, but in the adequacy of the methods employed. In the scratch test it is better to use pure dry pollens mixed with a drop of decinormal soda than ready-made extracts. If reactions are absent or doubtful by this method, intradermal testing with relatively strong extracts should be carried out.

2. Testing with dust extract from the patient's home or workplace in many asthmatics and in some cases of rhinorrhœa is important.

3. The treatment of hay fever without inclusion in the therapeutic extract of locally prevalent pollens to which the patient may be strongly sensitive, will not achieve the best results. Naturally the grasses which provide most atmospheric pollen differ greatly in various parts of Australia.

4. Treatment by specific desensitization cannot have much success without scientific adaptation of the dosage to the particular case, and continuation of inoculations until retesting indicates a considerable reduction of sensitivity. This sometimes requires much larger doses (the use of stronger extracts) than those which appear to have been employed by most workers in Australia.

5. The intradermal testing by autogenous bacterial cultures for indications of sensitiveness has been mentioned, together with an illustration of extreme susceptibility, necessitating relatively minute dosage in commencing treatment.

6. Attention has been drawn to the part played by other factors in disorders which have an allergic basis; and the necessity for dealing with these and for the general management of patients being treated by desensitization has been emphasized.

A CLINICAL STUDY OF MICTURITION.[1]

By E. GRAEME ROBERTSON, M.D. (Melbourne), M.R.C.P. (London),
Honorary Physician to Out-Patients, Royal Melbourne Hospital.

As the action of each of the two vesical sphincters and the relationship of their opening and closure to vesical contraction have not previously been studied, many theories of micturition have been elaborated upon an insufficient basis of fact.

Dr. Denny-Brown and I, in an endeavour to study disturbances of micturition consequent upon neurological lesions, elaborated a method of continuous recording of vesical and sphincteric action, and found it necessary to investigate normal micturition,(1) micturition after lesions of the *cauda equina* and after complete transverse lesions of the spinal cord.(2) It is with the results of the latter investigations, and deductions drawn therefrom, that this paper deals.

A few words may with advantage be spent upon description of the method. The patient's bladder is catheterized with a soft rubber catheter, lying in the lumen of which is a ureteric catheter. To each catheter is attached a separate pressure chamber, from which thick-walled rubber tubing leads to a tambour, on the rubber membrane of which is fixed a small mirror of stainless steel. A beam of light is reflected therefrom to a moving strip of bromide paper, and so continuous records are obtained. A side opening on each chamber affords means of filling the bladder or evacuating fluid through each catheter. A reservoir containing warm normal saline solution is attached to the pressure chamber of the outer catheter. The level of the pubic symphysis is taken, for convenience, as that of zero pressure, and by raising or lowering the reservoir the bladder can be filled or emptied, the small inner catheter meanwhile giving a continuous record of intravesical pressure. When the inflow is closed, vesical pressure is, of course, also recorded through the outer catheter.

A rubber balloon inserted into the rectum records intrarectal pressure, by reflection of a beam of light from its own tambour. Similar balloons bandaged to the abdominal wall and perineum record movement of these parts.

The bladder is first emptied and then filling is commenced, our usual procedure being to maintain the level of fluid in the reservoir at a height of 50 centimetres above the symphysis. After each 100 cubic centimetres of fluid has entered the bladder the inflow is closed and the vesical pressure at constant volume is recorded before filling is recommenced.

Figure I is a graphical representation of vesical pressure in a normal person during filling to 725

[1] This paper, which is based on clinical investigations performed with Dr. D. E. Denny-Brown at the National Hospital for Nervous Diseases, Queen Square, London, was read at a meeting of the Victorian Branch of the British Medical Association on August 7, 1935.

cubic centimetres. The vesical pressure gradually rises, but remains below 10 centimetres of water during filling to 500 cubic centimetres. At higher volumes the pressure rises more rapidly. When the inflow ceases after each increment of 100 cubic centimetres, the vesical pressure falls, and such a process of time-consuming adaptation to contained volume is found to lower the vesical pressure to a value similar to that observed during passive emptying, when the bladder is behaving more or less as an elastic sac.

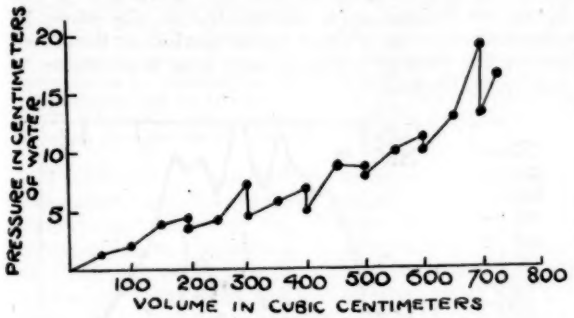

FIGURE I.

Graph of the relationship of volume of vesical contents to intravesical pressure during distension in a normal subject.

It is evident, therefore, that distension normally induces vesical contraction — a continuous tonic reaction of the musculature—which subsides, when filling is discontinued, to a lower level. Increasing tension of the vesical wall induces an active tonic response: the more rapid the filling, the higher the rise of tonic resistance for equivalent volumes. The contraction of the vesical musculature as a response to stretch can be demonstrated in phasic as well as in tonic effect, as a "secondary contraction" following with definite latency upon a brief abdominal pressure, a cough, or even a sigh (Figure II). The

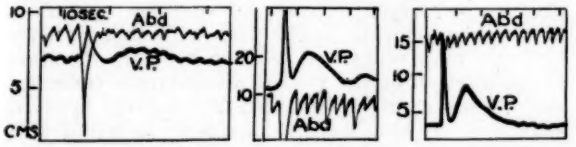

FIGURE IIA. FIGURE IIB. FIGURE IIC.

Tracing of records showing the vesical contraction stimulated by a brief passive increase in intravesical pressure. V.P. = vesical pressure, Abd = abdominal movement. In all tracings time is recorded in ten-second intervals by vertical strokes at the top of the record. The vertical scale represents vesical pressure in centimetres of water. In all tracings some records have been omitted and some vertically displaced to facilitate reading. Figure IIA shows a vesical contraction consequent upon a sigh in a normal subject (volume 150 cubic centimetres). Figures IIB and IIC record the response to brief passive abdominal pressures in subjects with *cauda equina* and complete transverse lesions of the spinal cord respectively.

reaction to distension may exhibit fluctuations of similar type to the last-mentioned secondary contraction. During distension small spontaneous fluctuations of vesical pressure (Figure IIIA, A and B) may occur at varying volumes, as, for example, during filling to 100 cubic centimetres and between volumes of 400 cubic centimetres and 500 cubic centimetres, and after cessation of inflow slowly dying away. When the volume had reached 725 cubic centimetres (Figure IIIB) the pressure rose steeply to a crest pressure of 48 centimetres. This was accompanied by a sensation of increasing discomfort, so that the subject immediately exerted an effort to hold water, and thereupon the wave rapidly declined. His relief was, however, but short-lived, for, upon relaxation of the effort to restrain imminent micturition, a long-continued wave-like disturbance reaching a high pressure developed.

FIGURE IIIA. FIGURE IIIB.

Figure IIIA: Tracing of record of vesical pressure in a normal subject, showing small spontaneous waves of vesical contraction (A and B) occurring during distension at a volume of approximately 625 cubic centimetres. Figure IIIB shows the final reaction to distension, in spite of powerful effort to restrain micturition, at a volume of 725 cubic centimetres. At C the outflow is opened and fluid also commences to escape around the catheter. The disturbance subsided only after a large volume had been expelled from the bladder.

Though at first modified by will, it proceeded to violent and painful vesical contraction, which subsided only when a considerable volume had been expelled from the bladder.

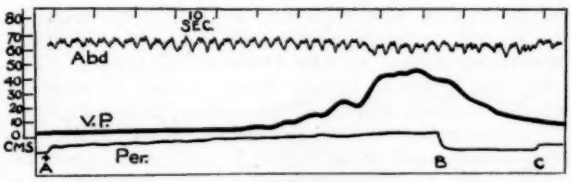

FIGURE IV.

Tracing of record of the attempt of a normal subject to facilitate micturition. Volume 200 cubic centimetres. Abd. = abdominal movement, V.P. = vesical pressure, Per. = perineal movement. At A the effort to micturate is commenced. At B the subject attempts to prevent impending micturition, and at C relaxes.

It was found possible to facilitate such vesical contractions by willed effort. At A in Figure IV the subject attempts to encourage micturition—the perineal musculature relaxes, the abdominal pressure remains constant. After a latent period, which in early experiments was of considerable duration, the vesical pressure commences to rise and does so in a series of superimposed waves which become more or less fused to form a crest. If the subject determines to "hold hard" (as at B), the perineal musculature abruptly contracts, and after a short latent period the vesical pressure commences to fall until it reaches its previous value. In such instances compounding of rhythmical waves of vesical contraction was evident, but with the more rapid development of high pressure responses fusion of such waves into a steeply mounting response occurred (Figures V and VI). A partial effort to hold water had the effect of dissecting out a rhythmical substratum from a powerful vesical contraction.

Because of these and other observations we have postulated the cortical control of micturition to be one of inhibition, unconsciously performed at low vesical volumes. As distension continues, higher fluctuations occur, sensations connected therewith reach consciousness, and inhibition becomes a conscious act, at first intermittent, later more constant. The high pressure responses we assume to be due to coordinated synchronous outbursts of nervous discharges from the sacral segments of the cord, a reflex response to stimulation of afferents in the vesical wall, the pathway lying in the pelvic nerves. The impact of the inhibition is assumed to be upon the neurones in the sacral segments of the cord, the latent period being due to the inevitable slow contraction of smooth muscle in response to a volley of impulses which have already left the spinal cord. Theoretically it is thus unnecessary to evoke inhibitory action of the hypogastric nerves. In this manner coordinated vesical contractions in response to distension are delayed until, upon relaxation of inhibition, the lower reflex mechanism of reaction to distension is released and a coordinated nervous discharge occurs from the neurones of the sacral part of the spinal cord. Simple contractions due to activity of peripheral plexuses may occur throughout filling.

In order to analyse the mechanism of the sphincters, a leak is now allowed through the side opening of the pressure chamber of the outer catheter, and this catheter is withdrawn until it no longer registers vesical pressure, its tip lying within or distal to the internal sphincter. The ureteric catheter, having been further introduced at the commencement of these manipulations, continues to record vesical pressure. If micturition be now commenced, as in Figure V, the vesical pressure rises, and, as the pressure mounts, the sphincter

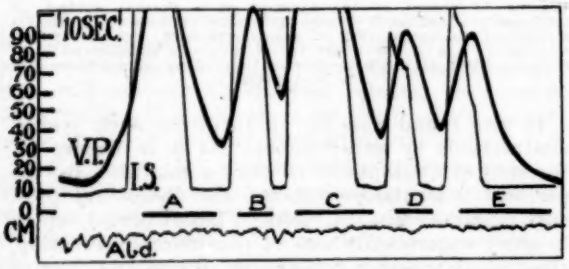

FIGURE V.

Tracing of vesical pressure (V.P.) and discharge (I.S.), in this instance through the internal sphincter. Normal subject. Initial volume 135 cubic centimetres. The duration of attempts to restrain micturition indicated by A, B, C, D and E. After each restraint the vesical pressure commences to fall and the sphincter closes in delayed manner, relaxing with the renewal of vesical contraction.

opens and discharge commences. As the vesical pressure falls at the spontaneous cessation of the act, the sphincter suddenly closes. If, during the progress of the contraction wave, the subject determines to hold hard, to prevent micturition (Figure V, A, B, C, D, E), the wave with short latency commences to subside, and only when the vesical pressure has reached a low level, some seconds after the onset of the restraint, does the sphincter close. Its opening and closure therefore apparently depend upon the accresence and subsidence of vesical contraction.

If now the urethral catheter be still further withdrawn, it is found that the curves of opening and closure are more abrupt and at somewhat higher pressures, and an effort to restrain micturition, when the latter is in progress, causes a sudden and immediate cessation of outflow (Figure VI). At the same time the perineal musculature contracts. If the effort to hold be relaxed immediately, the sphincter swings open widely, but if the effort be maintained, after a short latent period as described above, the vesical pressure falls and micturition is not reestablished.

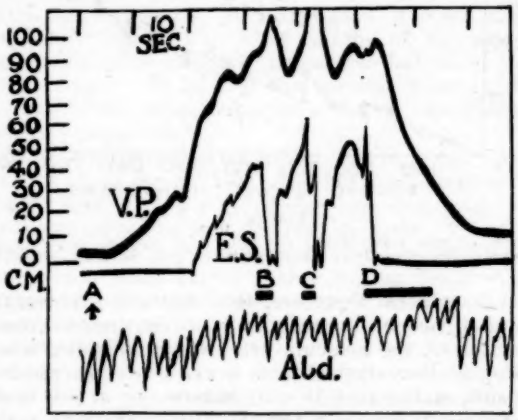

FIGURE VI.

The urethral catheter occupies a more distal position than in Figure V and here records discharge through both sphincters (E.S.). V.P. = record of vesical pressure, Abd. = abdominal movement. Normal subject. Initial volume approximately 100 cubic centimetres. At A micturition is initiated. B, C and D record duration of restraints, each causing abrupt cessation of discharge.

The immediate nature of this closure suggests the activity of striped muscle—the external sphincter. A proximal closing mechanism with long latency and inevitable reciprocal relationship with the state of contraction of the vesical musculature indicates the presence of a functionally active internal sphincter. If now the inner catheter be withdrawn until it ceases to communicate with the bladder, the action of the two sphincters can be recorded separately. The tip of the inner catheter lies between the two sphincters, that of the outer catheter beyond the external sphincter. Records taken with this disposition show that the external sphincter opens after the internal sphincter has relaxed sufficiently to allow fluid to enter the posterior part of the urethra and closes before the internal sphincter. Voluntary closure occurs only in the external sphincter, the internal remaining patent. The external sphincter is closed when micturition is in abeyance, for fluid introduced into the posterior part of the urethra, if under sufficient pressure, forces open the internal sphincter and enters the bladder. This procedure produces no sensation and has no influence in promoting micturition.

Powerful strains have no influence in promoting micturition; the vesical pressure at which the sphincter opens during vesical contraction is far below that at which it remains closed during a strain.

To quote, then, from our original paper:[1]

> The process of storage of urine and its evacuation occurs, therefore, in a reservoir, the distension of which excites a tendency to an automatic discharge. The primary factor in discharge is contraction of the wall of the bladder, with an accompanying reciprocal relaxation of the sphincters. The control of discharge depends upon conscious and unconscious inhibition of vesical contraction. Active voluntary excitation of the external sphincter may reinforce such inhibition. Usually the process pursues a course of progressive complete adaptation without the occurrence of sufficiently powerful vesical contractions to be accompanied by full relaxation of the internal sphincter until conscious and unconscious control of vesical contraction is released by voluntary effort.

Proceeding to pathological states, it may be remarked that the nervous supply of the involuntary musculature of the bladder wall and of the internal sphincter is from two regions of the spinal cord. One innervation is derived from the eleventh and twelfth thoracic and first lumbar roots and corresponding segments of the cord via the lumbar sympathetic chain, the inferior mesenteric ganglion and hypogastric plexus; the other, the parasympathetic outflow, from the second, third and fourth sacral segments via the pelvic nerves. These two outflows intermingle in the vesical plexus of ganglion cells and fibres, which innervate the bladder and sphincter. The external sphincter and associated voluntary musculature of the perineum are innervated from the same sacral segments, by the pudic nerves. All three sources of innervation contain afferent nerves.

To deal first with lesions of the *cauda equina* or *conus medullaris* destroying the second, third and fourth sacral roots or corresponding segments of the cord. Clinically the patient complains of the frequent onset of an unpleasant sensation, which he describes as a desire to pass water. To relieve this he strains and may secure evacuation of urine. If the straining process is not made sufficiently often, urine passes spontaneously, the patient is unable to prevent its passage, and there is no sensory experience beyond relief of the unpleasant, poorly localized sensation referred to above.

During filling the pressure mounts rapidly and may reach much higher pressures at small volumes than in the normal, for example, 70 centimetres at a volume of 200 cubic centimetres (Figure VII). The power of adaptation is highly developed. The more rapid the filling, the higher is the rise of tonic resistance for equivalent volumes. Simple waves of contraction may emerge from the tonic resistance and may be associated with discomfort. These spontaneous waves are each of the same duration as the response to brief stretch, and are regarded as single contractions of the bladder, the response to simultaneous stimulation of a large number of muscle fibres. Longer, more prolonged waves may result from compounding of several such small waves, but such compounding may be completely absent, and in *cauda equina* lesions is always slight.

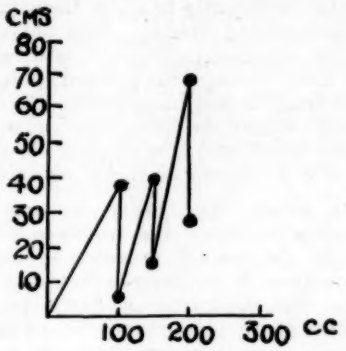

FIGURE VII.

Graph of relationship of volume of vesical contents to their pressure in a patient with a lesion of the *cauda equina*.

With the outer catheter withdrawn into the urethra it is found that intermittent spontaneous micturition accompanies these fluctuations when they reach sufficient amplitude (Figure VIII, S),

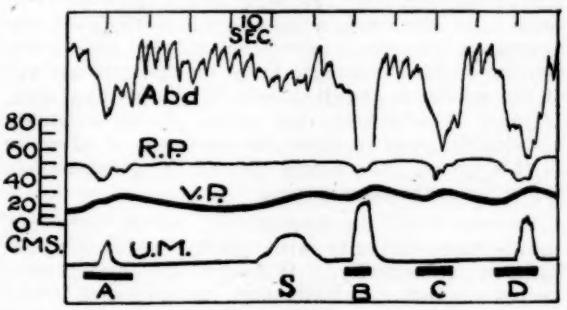

FIGURE VIII.

Tracing of a record of vesical pressure (V.P.) and urethral discharge (U.M.) in a patient with a complete lesion of the *cauda equina*. Abd. = abdominal movement, R.P. = rectal pressure. At A, B, C and D the subject "strains down"; at S spontaneous micturition occurs. Initial volume approximately 250 cubic centimetres.

but because compounding is slight the discharges are of small volume and occur frequently. They are often accompanied by an unpleasant sensation which causes the patient to assist discharge by straining. The curve of sphincteric opening and closure is symmetrically disposed in relationship to the vesical waves. Voluntary effort to prevent outflow is quite ineffective, the external sphincter being deprived of its nerve supply and paralysed.

As noted above, these patients have the power of expelling urine by straining. The intravesical pressure at which the sphincter opens varies greatly from strain to strain (Figure VIII, A, B, D), and sometimes a very powerful strain fails to extrude urine (Figure VIII, C). In the study of more extensive records of several patients it is apparent that the higher the vesical pressure before the effort, the lower the pressure at which the sphincter opens during the effort. Considered in terms of their inevitable reciprocal relationship, this means

that when the vesical musculature is in contraction, be it phasic or tonic, the sphincter is relaxed to an exactly corresponding degree. If the sphincter is about to relax sufficiently to allow discharge of urine from the contracting bladder, it is obvious that a slight passive increase in pressure will be sufficient to extrude urine through the relaxed sphincter. The discharge usually ceases immediately after cessation of the strain, unless the latter has caused a secondary vesical contraction, when the closure of the sphincter will be correspondingly delayed.

In other words, the ability to pass urine by straining depends upon the presence of detrusor activity. If the vesical pressure is low, a high passive pressure is without effect in provoking discharge. The discharge produced by straining falls far short of complete evacuation of the bladder. So to repeat: spontaneous detrusor contraction, both in the form of waves and maintained tonic contraction, is accompanied by laxity of the sphincter. If of sufficient degree, discharge occurs, and such discharge may be assisted by straining.

Regular waves of contraction during distension, temporal summation of such waves, and reciprocal relaxation of the sphincter imply some measure of nervous correlation. We believe that these contractions have their origin in peripheral plexuses, the nervous discharge being correlated by connecting neurones which dominate large numbers, if not all, of the scattered ganglion cells in the bladder wall. It must be admitted that there are no available histological data to prove the presence of afferents in this system; the complexity of the plexuses has defied accurate examination.

Intactness of the hypogastric nerves does not endow these patients with ability to inhibit micturition voluntarily. Whether the hypogastric nerves are intact or not makes no difference in the state of micturition, so it is unlikely that the vesical contractions are mediated by these nerves. This is borne out by animal experiments, for the same type of micturition develops when all the peripheral nerves are cut, or the lower portion of the spinal cord, including the origin of the hypogastric nerves, is destroyed.

Records of patients with transverse lesions of the spinal cord at any level above the sacral segments present a marked contrast. During filling the vesical pressure rises steadily, but only moderately steeply, until with suddenness, usually at a relatively low volume, powerful prolonged contraction reaching high pressure develops and, after lasting thirty to sixty seconds, subsides, only to recur in regular series (Figure IX). If the volume is kept constant, the large disturbance settles down in a series of diminishing contractions. If filling is recommenced, a similar increment causes another disturbance which, after subsidence, may again be stimulated by further addition of a similar quantity. These we suppose to be maximal responses and are stimulated only by increasing distension of the bladder musculature—increment of stretch. They may be associated with rectal contraction.

Abdominal pressure stimulates single secondary contractions (Figure IIc). Repeated abdominal pressures may stimulate maximal responses, with

FIGURE IX.

Tracing of vesical pressure during distension with 100 cubic centimetres of fluid in a patient with a complete transverse lesion of the spinal cord. When 100 cubic centimetres have entered, the inflow is ceased.

discharge through the sphincters (Figure X). During distension single spontaneous waves may occur, but are dominated by phases of explosive maximal contraction of compound nature, which show a tendency to repetition in slow rhythmic series. These fluctuations express the maximal correlation of summation of muscle fibre contractions. The production of these active contractions depends upon the intactness of reflex arcs through the sacral segments of the spinal cord. At a certain stage of summation active contractions produced by the local vesical ganglion cells become self-stimulating. A fresh variety of afferent nerve

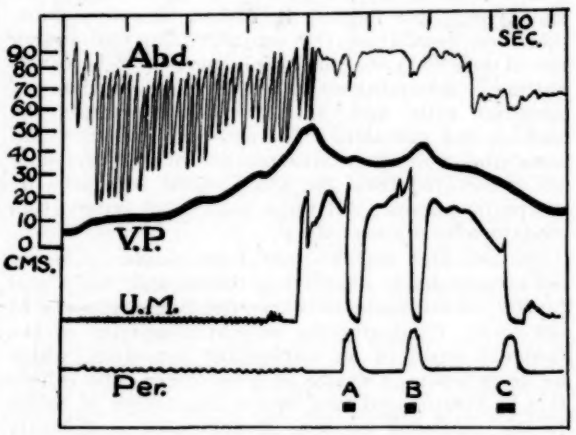

FIGURE X.

Micturition initiated by prolonged abdominal massage and interrupted by a series of scratches (A, B and C) to the sole of the foot in a patient with a complete transverse lesion of the spinal cord. Tracings as indicated in previous figures. Plantar stimulation causes contraction of the muscles of the pelvic diaphragm and the external sphincter, with interruption of the escaping stream.

ending, with connexions with the sacral segments of the cord, commences activity at a certain threshold of vesical contraction and evokes a correlated discharge of the whole efferent mechanism, sometimes including rectal activity. It is pertinent to remark that this maximal response differs in no way from that occurring in the normal when inhibition is no longer possible, or when micturition is allowed to proceed voluntarily. The difference, therefore, between normal micturition and micturition after a transverse lesion of the spinal cord lies in the presence of voluntary inhibition in the former condition.

When sphincteric activity is recorded, the internal sphincter is found to be acting exactly as in all other conditions: in true reciprocation with contraction of the detrusor. The external sphincter, while relaxing secondarily to vesical contraction and relaxation of the internal sphincter, shows also momentary contractions in response to plantar stimulation (Figure X) and involuntary flexor spasms. Continued stimulation is necessary to produce prolonged arrest of discharge, and if closure is prolonged and vesical pressure has fallen low, renewal of discharge may not occur. Contraction of the external sphincter and an inhibitory effect on vesical contraction are therefore a part of the flexion reflex. Head and Riddoch[3] described micturition occurring as part of the "mass reflex", depending upon "a vast outburst of motor energy overflowing into visceral channels". We have shown that on plantar stimulation the external sphincter contracts and thus micturition is effectively prevented during the reflex. Jerky abdominal contraction is a part of the reflex response if the lesion be high enough, and stimulation of vesical contraction by this means may lead to expulsion of urine after a definite latent period. So micturition, if it occurs, is secondary to the abdominal contraction which is part of the flexion reflex; if the abdominal muscles do not contract, micturition does not occur. When the secral segments alone are isolated, we have shown that both inhibition and less powerful facilitation of micturition occur, the latter with its focus at the meatus. For details of this the original papers should be consulted. After a transverse lesion of the spinal cord, voluntary contraction of the external sphincter is, of course, impossible. The external sphincter is always closed, and opens only in response to vesical contraction. Once relaxed in the course of micturition, it can be immediately and completely closed by peripheral stimulation and spontaneous flexor spasms. If the lesion be below the first lumbar segment, intactness of the hypogastrics leads to no voluntary control and, apart from conveying sensory fibres (which do not subserve normal desire for micturition) they are apparently little concerned with the function of micturition. Certainly they are not available for willed micturition, and there is no difference in the state of the bladder, whether the level of the lesion is above or below the origin of the hypogastric nerves. This has important bearing upon the rationale of the operation of so-called presacral neurectomy.

After a transverse lesion of the spinal cord, in the period of spinal shock, retention of urine occurs. The detrusor musculature falls into toneless inactivity, and the internal sphincter, in inevitable reciprocal relationship mediated by peripheral ganglion cells, falls into uninterrupted contraction. At this stage manual expression of urine will be difficult, even dangerous, and it is the first duty of the medical attendant to institute immediate means for regular emptying of the bladder. Intermittent catheterization at regular intervals is better than the use of an indwelling catheter, on account of production of a urethritis with the latter. However, continuous irrigation of the bladder with tidal drainage may more than compensate for the continued presence of the catheter. Suprapubic drainage has its advocates. In all cases avoidance of vesical infection is of extreme importance.

Further development awaits the recovery of detrusor activity, and this is the central fact in recovery from retention. An increasing tonic resistance and simple fluctuations of pressure occur as a response to distension, and with these the tone of the sphincter fluctuates, but in inverse manner. In the next stage, comparable with the permanent state after *cauda equina* lesions, the vesical fluctuations may reach a sufficient degree of peripheral coordination to evacuate urine through the correspondingly relaxed sphincter, and small micturitions occur. In the last stage of recovery maximal vesical contractions, reflexly stimulated by afferent impulses dependent upon the peripheral vesical mechanism, develop as the sacral segments of the spinal cord emerge from the depression of spinal shock. So complete automatic micturition, stimulated by stretch of the vesical wall, develops. The war-time observations of Gordon Holmes[4] upon recent spinal injuries traced the progressive recovery of vesical contraction from the toneless state in spinal shock. Our observations were made only in long-standing lesions.

It is pertinent here to remark that our investigations of rectal function, published in the current number of *Brain*,[5] allow of parallel deduction with but minor exceptions, for example, that voluntary control of defæcation depends solely upon the external sphincter. In conclusion, it may be stressed once again that the contractile power of the detrusor is the essential factor for micturition to occur. Given that, nothing else matters; failing that, nothing will work—micturition is in abeyance.

References.

(1) D. Denny-Brown and E. Graeme Robertson: "On the Physiology of Micturition", *Brain*, Volume LVI, 1933, page 149.
(2) D. Denny-Brown and E. Graeme Robertson: "The State of the Bladder and its Sphincters in Complete Transverse Lesions of the Spinal Cord and Cauda Equina", *Brain*, Volume LVI, 1933, page 397.
(3) H. Head and G. Riddoch: "The Automatic Bladder, Excessive Sweating and Some Other Reflex Conditions in Gross Injuries of the Spinal Cord", *Brain*, Volume XL, 1917, page 188.
(4) G. Holmes: "Observations upon the Paralysed Bladder", *Brain*, Volume LVI, 1933, page 383.
(5) D. Denny-Brown and E. Graeme Robertson: "An Investigation of the Nervous Control of Defæcation", *Brain*, Volume LVIII, 1935, page 256.

PROBLEMS OF JUNIOR RESIDENT MEDICAL OFFICERS.

By ISADORE IRVINE BRODSKY, M.B. (Sydney),
Resident Medical Officer, Prince Henry Hospital, Sydney.

RECENT developments strengthen the contention that junior resident medical officers have a definite claim for the investigation and rectification of:

(i) the conditions governing the appointments to the teaching hospitals on graduation, (ii) the type of work allotted to them during their period of residency, (iii) the amount of remuneration, (iv) the fact that, owing to the lack of cooperation, there is an absence of convenience both for doctors and hospitals in the filling of vacant positions.

Belated notice is being drawn to this neglected aspect of hospital administration in a variety of ways. Examples include: the failure of Adelaide and local hospitals to secure sufficient junior resident medical officers; statements made at the meeting of the Federal Council of the British Medical Association in Australia, the proceedings of which were reported in this journal,[1] where the opinions were advanced "that the work of a resident medical officer was not made sufficiently attractive for him" and that "resident medical officers were not sufficiently well paid".

The threefold purpose of this contribution is to analyse the position, to suggest improvements, and to bring the matter before the notice of the University of Sydney and the hospital administrators.

How are the appointments made? Before the final examinations students are asked to state, in order of preference, the hospital to which they wish to be appointed. After the pass and honours lists have been made available, the allocation of resident medical officers is carried out in accordance with an inter-hospital agreement, determined some years ago, with the concurrence of the University. The University will not supply the details of the agreement, but the appointments are made in the following way.

Those who secure honours at graduation are given a free choice of hospital. Alternately the hospitals then choose from the remainder, this being done, as far as is compatible, with the preference stated by the graduate. Sydney and Saint Vincent's Hospitals will not accept women residents, and this introduces an unnecessary complication to the system, throws a heavy burden on to Prince Alfred, and initiates a series of difficulties for graduate and hospital.

A graduate naturally is disappointed when he is allotted to a hospital which is not his first choice and which usually is not the hospital at which he has had his clinical training. Apart from sentimental reasons, not easily dismissed, a graduate may be expected to give more satisfactory service at a hospital with the routine of which he is more or less conversant. From any standpoint it is a mistake to permit any other arrangement. No athletic coach, after training a sprinter for three years, would ask him to start off the wrong foot. And the parallel does not wobble! The only possible objection that may be brought forward is that all trainees cannot be absorbed in the three teaching hospitals. But this objection is partly met by those who advocate the employment of more resident medical officers and the allocation of less patients to them. The whole problem is at present being tackled by a committee of the Faculty of Medicine, but as yet it has to secure unanimity of opinion between the hospital executives.

Next for consideration is the question of the type of work offered. At the Royal Prince Alfred Hospital the year's work is divided into four unequal sections, whereas five changes at intervals of ten weeks are made at Sydney Hospital. Each system combines advantage and disadvantage. Longer terms insure a more thorough acquaintance with the patients being treated and the therapeutic measures adopted. On the other hand, the Sydney Hospital scheme offers contact with a wider variety of patients. Let us examine the position at each hospital separately.

The junior receives an appointment at Sydney Hospital to work in medical, surgical, casualty and special wards, with a duplication of one of these. The difficulties encountered, and which are peculiar to this institution, are:

1. Honorary medical officers' patients are widely scattered over many wards (often six or seven), with a consequent waste of valuable time.

2. Juniors are given a week's notice or less of their new term's work, and they are thus deprived of any opportunity of reading up the work to be taken over.

3. The term's work which has to be duplicated is allotted irrespective of the wishes of the men, who may have a strong (and possibly easily met) preference for a particular section of the hospital work.

4. The work may be allotted in such a way that a junior may possibly not have any experience with general anæsthetics for the first six months (actual case).

5. Generally, juniors are second assistants at operations.

The Royal Prince Alfred Hospital's arrangements present deficiencies:

1. The top three graduates do not receive a general surgical appointment, their allotment being medicine, gynæcology, ear, nose and throat, and casualty.

2. There is no provision for holiday leave during the year, payment (approximately £3 8s.) being given instead, at the end of a year's service.

One important difficulty common to both hospitals lies in the extremely limited major surgical work handed to juniors. Here are figures and facts for the period January-September, 1935.

Royal Prince Alfred Hospital (19 resident medical officers): 52 appendicectomies, one herniotomy.

Sydney Hospital (15 resident medical officers): 35 appendicectomies, four herniotomies.

The average, as can be seen, for each hospital is less than three major operations for each resident

medical officer. Eight resident medical officers have failed to secure any work of this kind, while four of the Royal Prince Alfred Hospital resident officers between them are responsible for three-quarters of the total list. It is interesting and very instructive to note that the resident medical officers attached to the professorial surgical unit have a combined tally of 22. By unfavourable contrast, 17 other resident medical officers have shared 15 appendicectomies. The Sydney Hospital figures on analysis show that three men have been given 21 cases, which makes a poor average for the remaining juniors. No attempt can be made to estimate the number of cases offered to juniors without being solicited. From the viewpoint of this article it would be useful to know how many cases came from senior and junior honorary medical officers.

Various reasons are given in support of the parsimonious policy. One is convincing: juniors have had no or very little experience. Is this lack of experience to tie their surgically inclined hands for all time? When are they to get the experience? And what of Hazlitt's observation that success prompts to exertion and that habit facilitates success?

Obviously the time and place to acquire this experience is during the surgical term's work. Juniors enter the teaching hospitals to gain experience under the optimum conditions, and expect at the end of twelve months to be able to cope independently with an average appendicectomy at least. They are fully aware of their limitations. They have been taught and forced to realize that surgical skill and confidence have a foundation in practice, experience and judgement.

It should be the aim of the hospitals to equip their graduate trainees for the task of initiating and completing some of the common surgical procedures. Opportunities must be provided without any solicitation. Senior and junior honorary medical officers should cooperate in providing work for the senior and junior resident officers. The lead must come from the top. Let it come in the form of increased opportunities, advice and supervision.

Surgeons should contrast their ungenerous attitude with that of the physicians, and make some effort to "make the work of resident medical officers more attractive by giving them a proper amount of responsibility".[1]

Another, and vexed, question is that concerning the status of resident medical officers. It is often forgotten that they are doctors. Sometimes it is doctors themselves who forget this. Surely the much-vaunted freemasonry of medicine should operate to better effect. In general, a junior does his best to work harmoniously with his environment, medical and lay. But there is a tendency among some senior members of nursing and technical staffs to belittle, openly and covertly, the knowledge of juniors. Junior resident medical officers are entitled to the fullest protection against this, otherwise their part in team work may be less efficient and their position in the hospital apt to be undermined. Juniors are professional men. They should be given professional work; they should command and be accorded professional respect.

In short, juniors enter teaching hospitals, particularly Sydney Hospital and the Royal Prince Alfred Hospital: (i) in the hope of leavening the dough of theory with the yeast of practice, and because of the association with and teaching of the more experienced members of the profession; (ii) because they consider it gives them added status, which may be of value in securing appointments here and overseas at a later date; (iii) to carry out professional tasks and to fit themselves for general practice or for further post-graduate work.

Remuneration.

By definition[2] an honorarium is a fee for professional services, and a salary is fixed periodical payment made to a person doing other than manual or mechanical work. Hospital officials maintain that juniors receive an honorarium, in the sense that the amount paid is not to be considered as payment for work done. No useful purpose can be served in attempting to distinguish between the words. Getting down to essentials, juniors receive approximately £1 12s. 6d. a week. At the end of a year's service they receive a bonus of £20 (less tax) at the hospital's discretion. Should they leave before completing their time, they forfeit the bonus. The Royal Prince Alfred Hospital insists that its juniors stay twelve months, and requires a contract to be signed to that effect. This system has not yet been adopted at the Sydney Hospital, but the men have been warned of various consequences. These include the loss of the bonus.

Juniors should earn sufficient money to confer on them a fair measure of independence. Certainly, when they reach the average age of twenty-four, their parents should be freed from a responsibility which, under this set of conditions, properly belongs to the hospital. All concerned should realize that the money paid is a concrete recognition of this responsibility, and that money is never intended to recompense for interest in work. Advantage need not be taken of the fact that juniors are prepared to make a financial sacrifice. It is most difficult to reconcile the financial treatment of juniors with that received by other members of hospital staffs, which are laden with untrained and unskilled employees who receive greater emolument for relatively inferior performances over shorter working time.

In view of the fact that juniors are the vital medical link between patient and honorary medical officer, and actually shoulder a lot of responsibility in the care of the patient, the claim for an increased reward is manifest. Making the ample allowance of £2 a week for the accommodation, food and laundry

at present provided, the weekly amount should, together with the bonus, be equivalent to £5. The holiday allowance should be £10 for two weeks, instead of at the present rate, which discards the fact that the hospital services are not utilized during this period.

Hospital Changes.

Lastly, there is the matter of changes made from one hospital to another. Before the termination of the year, resident medical officers have weighed the advantages of senior residency, obstetric or pædiatric or general hospital appointments. Because there is no coordinating organization, vacancies occur during every month of the year. Competition is keen for these vacancies, and when these arise in the larger hospitals supply more than equals demand. In order to eliminate some of the competition, juniors, suiting their own career and convenience, often transfer their services within six months or so of their first appointment. Unfortunately this disjoints hospital routine. Is the junior to shut his eyes to beckoning opportunity? Hospitals say yes. No is the correct answer. And the remedy? Adjust all appointments, by inter-hospital agreement, so that vacancies fall due in January for preference, and/or July. Changes made at these times, and in the way suggested, will be more suitable for the resident medical officers and, at the same time, will be less likely to disrupt hospital organization.

Two bodies exist which could have done a great deal in these matters. Reference is made to the Royal Prince Alfred Hospital Residents and Ex-Residents' Association and the Old Sydney Hospitallers' Club. Something attempted, something done, sums up the position.

By forming a Resident Medical Officers' Association of New South Wales, resident medical officers could satisfactorily achieve what have been ill-defined objectives. An organization of this type could cater for senior resident medical officers too.

Conclusion.

A plea has been made for investigation and correction of the conditions under which junior resident medical officers work, particular attention being focused on the method of appointment, type of work, amount of remuneration, and facility for inter-hospital changes.

The need for the supply of extra surgical work is stressed; also a plea is made for an increased recognition of the status of junior residents.

It is suggested that a resident medical officers' association of New South Wales be formed.

References.

(1) Meeting of Federal Council, THE MEDICAL JOURNAL OF AUSTRALIA, September 28, 1935, page 424.
(2) The Concise Oxford English Dictionary, 1934.

Reviews.

ENDOCRINOLOGY.

THE field of endocrines is now so wide that it requires an author who possesses wide personal experience on the subject to do it justice; otherwise a text-book might resemble one of those anthologies that are so popular as Christmas presents. Herman Zondek's book on diseases of the endocrine glands is no mere summary of the work of others, and its third edition is worthy of more than passing notice.[1] This fuller and revised version of previous editions is a clearly written exposition of this most difficult subject, in spite of occasional infelicities on the part of the translator and a rather closely knit style. Perhaps one of the most valuable sections of the book is that summarizing the physiology and chemistry of the hormonal glands, a mere sixty pages out of a total of well nigh 450, but full of "meat". The clinical sections are also full of matter that is interesting and well arranged, and a liaison between laboratory and ward is established wherever it is possible. The author has wisely devoted more space to the commoner than to the rarer diseases, though some of the latter, such as generalized *osteitis fibrosa*, might perhaps have been dealt with in greater detail with benefit. Thyreoid disease is particularly well discussed. All may not agree with the author in his views, though his stressing of his own "peripheral" theory emphasizes the need for looking beyond the thyreoid gland in attempting to understand toxic goitre. Nor will his ideas of treatment entirely concord with those of all Australian practitioners; for example, with regard to the use of continued iodine therapy in some cases and the selection of cases for operation or radiation. But as he points out himself, there is a regional difference in thyreoid disease; this may well affect the views of those who have to treat it. With regard to treatment in general, throughout the book the Continental tendency to use proprietary preparations is often evident; it will be noticed also that Zondek uses not only the parenteral, but the oral preparations of some glands, such as the pituitary and ovary. In some of the doses recommended one might doubt the efficacy of the oral preparations, but perhaps after all it is not always possible to base the treatment of the individual on as rigid a scientific basis as is the understanding of the disease from which he suffers.

Endocrine obesity is described under several headings, according to the hormone supposedly affected, and due attention is paid to the importance of the diencephalic centres in connexion with the so-called pituitary types. Water-salt obesity is given special attention. Only scant attention is given to the dietetic side of treatment of obesity in general; but perhaps this is in keeping with the scope of the book. It is of interest that Zondek recognizes the "*status thymicolymphaticus*", though he does not attach special significance to the thymus. The sex gland disturbances are given rather less space than might be expected, but the information is compact, and due warnings are given against over-enthusiasm in treatment.

As a whole, the book is conservative and moderate in tone, and the author does not attempt to fit all the syndromes he describes into a definite endocrine pattern. It is excellently produced and copiously illustrated, and may be confidently recommended as a modern account of the diseases of the ductless glands; it does not attempt to be encyclopædic, but its full bibliography should supply the needs of those who require fuller detail. If it is stronger on the physiological and descriptive sides than it is on the therapeutic, this is after all only a reflex of our present knowledge of the subject.

[1] "The Diseases of the Endocrine Glands", by H. Zondek, M.D.; Third Edition, revised and enlarged, translated by C. Prausnitz, M.D., M.R.C.S., L.R.C.P.; 1935. London: Edward Arnold and Company. Super royal 8vo., pp. 503, with illustrations. Price: 40s. net.

The Medical Journal of Australia

SATURDAY, DECEMBER 28, 1935.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

ON A BIRTHDAY PRESENT.

To lessen pain and suffering, to save life in a world red with mass murder, and to earn the love of his colleagues—what better fate may befall a man? Such has been the lot of Sir Thomas Barlow, now in his ninety-first year. In the evening of his life a noted English journal[1] has issued a Birthday Number, bearing his name. It is a graceful gift to one who for years was among the leaders of British medicine. Sir Thomas Barlow has been a noted physician; but more especially has he made the study of sick children his sphere. It was he who, amongst the first of modern days, announced that rickets and infantile scurvy were distinct diseases, supporting his statement with pathological evidence which none but the blind might doubt; it was he who, amongst the first of his time, introduced the now accepted diet for the cure of the latter disease.

For centuries scurvy in the adult form was the foe of armies on land and the crews of ships at sea. It slew crusaders by the thousand, decimated the sailors of Columbus, Vasco da Gama, Cabot and Magellan, and played havoc amongst the seamen in the Eastern spice trade. As much as the hazards of the ocean, scurvy caused the almost complete destruction of the gallant Anson's expedition. Tough John Hawkins, dying of fever off Puerto Rico, said he had seen twenty thousand men die of scurvy during his twenty years afloat. Knowledge of a cure came slowly, sporadically, and in divers places. In 1535, an expedition under Jacques Cartier reached Labrador. The French sailors, scorbutic to a man, offered suppliant prayers before a crucifix set up on the beach. Prayers availed them nothing until friendly Indians healed their visitors with decoctions of spruce tree needles; "and that tree did more in six days to cure them than all the doctors of Louvain".

We know that sixteenth and seventeenth century writers saw scurvy as a whole—as one disease. But it was long before any doctor believed, in those days of enlarging horizons, that the ordinary stay-at-home might contract it. Even when the thing was known by unmistakable signs to occur in civilian populations, nobody seems to have noted its presence among infants. Yet there it must have been; a scorbutic mother in those days of universal suckling could have nourished none other than a scorbutic child.

In the days of Raleigh the potato (along with tobacco, chocolate, oranges and the papaw) was ferried into Europe, and for the next forty years was to be found only as a luxury on the tables of the very rich. "Grow potatoes!" said the Royal Society again and again; but not until the latter years of the eighteenth century was their price within the reach of the poor. Meantime, though scurvy was everywhere, its juvenile form remained undetected for the most part. Yet here and there were men with eyes and brains; Francis Glissan, in 1650, had written a clear and succinct description of the infantile form of the disease. He was forgotten, and his work was shelved—missed entirely, even by such acute clinicians as Heberden and Underwood. Some believed (and wrote) that the scurvy of adults was one with the rickets of children. In the fifties of last century, cases of undoubted infantile scurvy were labelled as "acute rickets".

[1] *Archives of Disease in Childhood*, August, 1935.

So the matter rested until Cheadle, a large-boned Yorkshireman—a physical giant of great attainments—appeared upon the stage. Cheadle had been saved for the service of medicine through the operation of the strange laws of Chance. Chosen to row for Cambridge in the boat race, he became ill at the last minute, and was unable to take his place. The boat sank during the race—and Cheadle could not swim! After graduation, he accompanied Lord Milton on a perilous enterprise. The two men, leaving Quebec in 1862, crossed the Rockies into British Columbia, blazing a new trail—the so-called "North-West Passage by Land"—from the Atlantic to the Pacific. During this dangerous journey Cheadle faced hunger and death and saw much of smallpox and, especially, of scurvy. Back in London, he soon became assistant physician to the Hospital for Sick Children, Great Ormond Street. There he saw many little patients obviously suffering from scurvy. The attending physicians, powerless to cure, were clipping away large fungating masses from the gums of their charges. In *The Lancet* of 1878 appears a clinical lecture by Cheadle, his account of three cases of scurvy supervening upon rickets. Cheadle is a figure in modern medicine; his hospital work took precedence of all else; he bade his hospital residents, all pleasures laid aside, to be on call whenever one of their patients was in peril. Dr. F. John Poynton's boast is that his own name appears beside that of Cheadle in the article on infantile scurvy in Allbutt and Rolleston's "System".

In 1883, Thomas Barlow, also assistant physician at Great Ormond Street, settled once and for all the scurvy-rickets relationship. The title of his paper was: "On Cases Described as Acute Rickets, which are Probably a Combination of Scurvy and Rickets, the Scurvy being an Essential and the Rickets a Variable Element". Barlow's paper proved his case to the hilt. It was, as some have said, a model of clarity, logical exposition, and intellectual probity; and it was supported by pathological evidence that put the whole matter beyond doubt.

Yet much remained and still remains to be done. What substance or substances in orange or meat juices, what magician lurking beneath the jacket of a potato worked the cure in infantile scurvy? This challenge to workers in carbohydrate chemistry was accepted. In these columns notes have appeared on the work, now twenty-three years old, of Holst and Fröhlich upon guinea-pigs and other mammals; already we have taken note of von Szent Györgyi's work upon the isolation of a crystalline substance, "hexuronic acid", from cabbages, oranges, and the adrenals of the ox. We have mentioned the work of Cox, Hurst, and Reynolds, and touched upon the reasons for the adoption of the name "ascorbic acid". All this and much more is to be read between the covers of the birthday gift to Sir Thomas Barlow. We hope that he looks back with pride on his distinguished career. He was amongst those who attended King Edward VII when, just prior to his coronation, that monarch underwent operation for appendicitis, and the nation palpitated between hope and fear. The story runs that during that anxious time neither Treves nor Barlow sought his bed for a week. Honour and ease are seldom bedfellows. Both have now come to Sir Thomas Barlow. He is a link in the historic chain which stretches back beyond the days when Columbus and his scorbutic crew, searching for pepper, blundered upon the West Indies.

Current Comment.

BREATHING EXERCISES IN ASTHMA.

JOHN FLOYER was a doctor who published, in 1698, his "Treatise of the Asthma". Himself an asthmatic, he described with an inner knowledge the "straitness, compression, or constriction of the bronchi" which occurred during his attacks; and he believed that certain weathers, improper food, some odours, and even an access of his own passions might send him wheezing to bed.

Floyer's striking picture of his sufferings prompts the reflexion that though asthma is an ancient disease, a fundamental knowledge of its causes and attempts at its scientific treatment are things of our own day. Not long ago an asthmatic patient was a neurotic patient. Our knowledge increased, however, when Schloss in 1912 related the story of a child in whom attacks were produced by

eating eggs, and when Goodale in 1914 drew attention to the nature of "horse" asthma. During the years since the War the hopes of the asthmatic have brightened, for the period has seen the rise of asthma clinics in many large hospitals. L. J. Witts,[1] for instance, set himself the huge task, covering four years, of making a critical survey of 500 patients attending Guy's Hospital, using as controls 100 persons with no symptoms of asthma. J. B. Christopherson[2] seems to have been amongst the first to advocate a closer anatomical and radiographic study of the lungs and air passages in cases of bronchial asthma and bronchitis, and to advance the claims of lipiodol as an adjunct in this research. Lack of space prevents more than mere allusion to such researches as those into the relation of asthma to the adrenaline content of the blood, to the observations of Oriel on urinary proteose, or to such new methods in treatment as the use of allergen-free chambers, intramucous autoserotherapy, and a number of recently introduced drugs.

From the clinic of King's College Hospital has appeared a report by James L. Livingstone and Marjorie Gillespie[3] dealing with the effects upon asthmatics of breathing exercises. It is a strange fact that asthma rarely affects professional singers, who must practise such exercises in the learning of their art. Teachers of singing, consequently, have advocated these methods as a means of controlling asthma; and in London, Miss Ella Seyfang has for many years been successful in this regard. The object of respiratory control, then, is the proper control of the diaphragm and the mobilization of the chest wall; to achieve these ends the physiotherapeutic department of King's College Hospital contrived a list of exercises, simple and readily learnt by patients. Livingstone and Gillespie have observed for twelve months the clinical course of 75 of these asthmatics, all of whom went through the prescribed routine. While the aim of the investigators was to test the effect of the exercises alone, they were hampered by the certainty that most hospital patients set great store by bottles of medicine; they had to swallow something, even if it were only burnt sugar. The observers therefore compromised by allowing the patients to take a simple mixture of iodides, paregoric, and stramonium, as well as some form of ephedrine, should serious attacks occur. All these drugs were little by little withdrawn as the patients under treatment acquired confidence and hope.

It is believed that during an attack of asthma the bronchioles narrow as the result of congestion and spasm. Strong inspirations draw in air, but this air is imprisoned in the alveoli. Thus, all asthmatics find difficulty with expiration. There is a resulting over-distension of the chest and a serious reduction in gaseous exchange. During the attacks, respiration is of the thoracic type, and the accessory muscles of breathing are strongly contracted. The diaphragm, depressed to its full extent, contracts spasmodically or not at all. The patient, as his dyspnœa increases, essays further inspirations, only to distend his chest still more. Gradually the characteristic deformities of the thorax appear—the pigeon chest, the Harrison's sulcus, the kyphosis. They are born of repeated spasm of the intercostal and accessory muscles, and of the diaphragm. The expansion, measured at the level of the fourth rib, may be no more than one inch.

It is therefore obvious that breathing exercises in asthma should have as their object the emptying of the lungs during the expiratory phase. Further, attempts must be made to secure normal diaphragmatic movements, relax muscular spasm, mobilize the ribs, and lessen kyphosis. Briefly, the patient is told to take a short inspiration through the nose, followed by a long expiration through the mouth, of such sort as to sound like a whistle or a hiss. During this latter phase the epigastrium must become hollow to permit of the rise of the diaphragm. Female patients must throw away their corsets, however new.

The tuition takes time; the teacher must patiently drill the patient twice or thrice a day for ten minutes at a stretch, must offer words of encouragement, and persevere until the subject acquires the ability to make normal diaphragmatic movements. As part of the usual routine, the excursions of the diaphragm are periodically inspected through a screen. For this purpose, or for other observations, the patients attend the hospital twice a week, each of them having made records of any asthmatic attacks during the interim. In order to gauge the full effect of the exercises, the observers disallowed the use of vaccines and the removal of sensitizing proteins; they also discouraged all intranasal operations, even when these seemed highly necessary.

As the fruit of a year's supervision along these lines, Livingstone and Gillespie maintain that half their patients (38 out of 75) are free of symptoms, 14 show most decided improvement, 12 have benefited, while 11 show no betterment in the asthmatic condition. It seems that happier results follow the treatment in allergic patients than in those of the bronchitic group, who suffer greatly during the winter. Amongst the women, improvement is greater than amongst the men, a probable reason being that the former normally are poor diaphragmatic breathers and so offer larger scope for favourable results under proper tuition. They derive considerable advantage, too, from the disuse of corsets. Length of years seems to have no adverse influence upon the results of the treatment; Livingstone and Gillespie were astounded at the increase in chest movements among some of their elderly patients. After a course of breathing exercises, a patient whose asthma is cured, or whose condition is greatly improved, fulfils the following criteria: the chest expansion at the level of the fourth rib is at least two and a half inches; the epigastric expansion is at least two inches; there is power to dissociate thoracic from abdominal

[1] *The Lancet*, February 10, 1934.
[2] *The American Journal of the Medical Sciences*, October, 1933.
[3] *The Lancet*, September 28, 1935.

breathing on the word given; there is complete control over the full range of the diaphragmatic movement.

The psychological aspect of this treatment is of great importance. It inspires confidence in the patient and teaches him to rely on himself and not upon doctors and drugs. It is to be hoped that these results of Livingstone and Gillespie will be corroborated by others and that they will not suggest a rich field for exploitation by imposters.

LYMPHOGRANULOMA INGUINALE.

LYMPHOGRANULOMA INGUINALE was first described by Chassaignac in 1859. Durand, Nicolas and Favre, in 1913, recognized it as an entity. It is definitely a venereal disease, and is transmitted only by direct contact. The ætiological factor is unknown, but is generally held to be a filter-passer. *Bacillus gangrænæ cutis*, allied to Ducrey's bacillus, has been considered the causative organism. The disease is known by a variety of names, such as "tropical" or "climatic" bubo. One of these names ("non-venereal" bubo) is at variance with the facts. The condition occurs frequently in seamen and is largely a tropical disorder, but cases have been reported arising in France and England. Diagnosis has to be made from chancroidal bubo, syphilitic adenitis, tuberculosis, Hodgkin's disease, malignant disease, and *pestis minor*. The evolution of the disease is very slow. The tongue, palate and neck have been attacked, but implication of this sort is very rare. In 1925 W. Frei introduced his intradermal inoculation test. Frei's antigen is made by aspirating the contents of a softened bubo which has not formed a fistula. This is diluted with normal saline solution and sterilized on two successive days. It is injected intradermally into the arm. In *lymphogranuloma inguinale* a marked papular reaction occurs in forty-eight hours. This reaction is not given by chancroid bubo or *granuloma inguinale*. A slight redness may appear in normal cases. However, Frei's test is not accepted by all observers as specific.

J. A. Bourgouin reports three cases and discusses the condition.[1] He points out that manifestations are remittent. The course is protracted and three stages are generally recognized. The local lesion is usually confined to the genitals, but rarely may be extragenital. Next follows chronic inflammatory implication of the adjacent and more remote lymph glands. The third stage exhibits lesions in non-lymphatic tissues, which are directly associated with the destruction of lymphatic glands and vessels. In the male the primary lesion occurs on the *glans penis* or in the balano-preputial sulcus. The time between exposure and the appearance of a primary sore is considered by some observers to be between ten and thirty days, but the period is difficult to estimate. The lesion is usually small and evanescent; it may be papular, nodular, or ulcerative. A lympho-granulomatous urethritis may develop. After about two weeks the superficial inguinal lymph glands enlarge. Elephantiasis of the penis and scrotum may follow lymphatic obstruction. The adenitis may be unilateral or bilateral. The glands, at first discrete, become matted together. The skin adheres to the underlying glands and becomes red and later violaceous in colour. The external iliac glands become involved. There may be no suppuration, but often areas of softening appear and multiple fistulæ form. Pain is usually absent, but tenderness over the glands occurs. Other manifestations may be pyrexia, frequent pulse, arthritis, and *erythema multiforme* or *nodosum*. A leucocytosis (up to 20,000) with increase of the large mononuclear cells is of diagnostic importance and implication of the deep iliac glands is deemed to be pathognomonic. The second stage may last for months, the glandular inflammation being replaced by fibrosis. In men the third stage is rare, appearing as rectal stricture. This stricture may be combined with pelvic cellulitis, fistulæ and absesses, or elephantiasis of the external parts.

In the female the primary sore is at the fourchette or in the vagina. In both sexes the primary sore may escape observation. In the female inguinal adenitis rarely suppurates and the only common manifestations are late, such as stricture of the rectum. Esthiomène (*ulcus vulvæ chronicum elephantiasticum*) is a chronic ulcer of the genito-anorectal region often attended with pudendal elephantiasis. Bourgouin investigated Frei's test, using, as a control, saline solution with 0·25% of phenol. He found that it was often necessary to use more than one antigen to obtain a positive reaction. Antigen loses much of its strength after six months. Bourgouin points out that R. Bensaude and A. Lambling insist that the result of Frei's test must not be read before the third or fourth day. The reaction may persist for ten days or longer. Not every case gives a positive result to the test. Cross-testing may be made. Antigen from a suspected case may be injected into a patient known to have the disease. If the patient gives a positive reaction, the suspected person may be deemed to have the disease. Cases are reported in which a person who did not react to the Wassermann test gave a positive reaction as a result of lymphogranulomatosis, without manifesting any evidence of syphilis. Again, some patients with lymphogranuloma, who have syphilitic lesions as well, fail to react to the Frei test, but give a positive reaction after antiluetic treatment. It is obvious that there are unexplained vagaries in the application of Frei's test which militate against its usefulness.

Cases of this disease should be looked for in the northern parts of Australia. Ulcerating granuloma of the pudenda has been recorded from those parts, and it is certainly possible that *lymphogranuloma inguinale* also occurs. Unfortunately treatment is very unsatisfactory.

[1] *The Canadian Medical Association Journal*, September, 1935.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE SOUTH AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Anatomy Theatre, University of Adelaide, on August 29, 1935, DR. D. R. W. COWAN, the President, in the chair.

Some Allergic Disorders.

DR. D. L. BARLOW read a paper entitled: "Some Allergic Disorders" (see page 883).

DR. L. W. LINN said that he would like to express his appreciation of Dr. Barlow's very comprehensive and interesting paper. As Dr. Barlow had mentioned, a tremendous amount of literature had grown up in the last few years concerning allergic diseases, and it was very difficult to keep a clear and rational outlook on this subject. Attempts had been made from time to time to include so many diseases of such diverse characters in this group that there was little wonder that so many regarded the whole subject with a certain amount of scepticism. He thought, therefore, that an attempt should be made to simplify the whole matter and to keep the clinical point of view in mind as far as possible. In 1906 von Pirquet coined the term "allergy", by which he meant "an altered reactivity", and this to Dr. Linn was still the simplest definition. Allergy was just an altered reaction or a different reaction from the normal of various groups of cells to certain foreign substances. As to what changes occurred as a result of this altered reaction, again aiming at simplicity, he regarded the essential pathological change in all allergic conditions as being an œdema, and he hesitated to include any condition under the heading of allergy unless it was quite clear that there was or had been an œdema of the tissues involved. He was well aware that there were other changes as well; that there might be a spasmodic contraction of unstriped muscle, if there was any present in the parts involved; that inflammation and even tissue destruction might follow; but if the condition was a true allergic one, there must be an exudation of serum. Simply, then, allergy was an altered reaction from the normal of certain structures, and the essential pathological lesion was an œdema of the parts involved. Although this definition might not satisfy all the scientific requirements, it made it possible to keep a clear outlook from a clinical point of view.

In the treatment of asthma Dr. Barlow had mentioned the so-called "heavy desensitization" adopted in some cases. Although many patients became symptom-free after three or four thousand units, a few were unrelieved even with ten thousand units. He was sure that in these instances results would be greatly improved if higher doses were used. Freeman and Bray had given up to one hundred thousand units in some cases. In giving these larger doses it was usually necessary to combine some adrenaline with the injection, about 0·25 cubic centimetre, to delay absorption and to counteract a general reaction. Even with the adrenaline added, he had twice seen a severe reaction, and it was very important when giving large doses to keep the patient under observation for at least half an hour after the injection. Freeman was very enthusiastic concerning vaccine therapy in asthma in the absence of positive reactions to pollens or epithelial inhalants, such as feathers, hairs, dust *et cetera*, and last year, instead of accepting for the vaccine the organism with the predominating growth on ordinary culture, he had made increasing use of a special method of vaccine preparation whereby the organisms to be incorporated in the vaccine were selected after the bactericidal power of the patient's own blood had been tested against the various organisms isolated from the patient; in other words, the organisms used in the vaccine were those which had withstood the bactericidal action of the blood. Often these organisms had been quite insignificant on ordinary culture, and spectacular results had been frequently obtained in this way when treatment with a vaccine made in the ordinary way had been quite ineffective.

As far as skin testing was concerned, the method of choice in London at present was the intradermal one for everything except pollens, and for pollens the scratch method and pure dry pollens were used. Although Dr. Linn had seen a very large number of patients tested intradermally, it had never been his misfortune to see any untoward symptoms, but cases of severe general reactions had been reported following this method, so his usual routine here was to perform the scratch tests first and then, if these gave no reaction or indeterminate results, to proceed with intradermal tests. In the interpretation of the tests he was not quite satisfied to call an erythema of one-quarter of an inch diameter a positive reaction, unless there was a slight wheal as well; an erythema might be suggestive, but he thought there should be some œdema present, even though it might be only very slight, in order to regard the reaction as a positive one.

Dr. Barlow had mentioned hay fever or spasmodic rhinorrhœa. It was true that hay fever was really a seasonal spasmodic rhinorrhœa, but Dr. Linn thought it was better to consider the two conditions separately. The cause of hay fever was usually fairly easily determined, but that of spasmodic rhinorrhœa, while generally a common inhalant, like feathers, danders *et cetera*, might be very difficult to discover. However, once the cause was found, treatment was usually equally effective in both conditions by means of desensitization, and Dr. Linn was convinced that this was the method of choice in treatment at the present time. In Germany there was a hay fever association and, incidentally, they held their annual conference in the middle of the hay fever season on the open, treeless island of Heligoland. The medical officer of this association, after an analysis of 2,000 cases, strongly recommended "rational therapy", as he called it—desensitization with the offending pollens—and he claimed that 90% of patients thus treated obtained complete or very nearly complete relief from symptoms.

As far as the manifestations of allergy in the skin were concerned, one could regard them all, eczema, urticaria *et cetera*, as being an altered cutaneous reaction. The simplest type of allergy was seen in the skin, and it could be studied much more readily histologically than in other parts. It was found that there was always an exudation of serum, but the clinical picture varied according to the situation of the sensitized cells and the nature and intensity of the irritant. If the reaction occurred in the epidermis, an eczema or dermatitis resulted; if in the corium, one got urticaria or perhaps *erythema multiforme*; and if in the subcutaneous region, angioneurotic œdema. As in all other allergic conditions, heredity, emotional factors, and so forth played an important part, but the toxin or irritant causing the reaction was by far the most important element. The toxins might be produced from numerous sources, both exogenous and endogenous, and be physical, chemical or bacterial. In treating these cases the objective was always to try to determine, if possible, the nature and source of the irritant causing the reaction. Skin testing was often very useful, but perhaps not quite so useful as in other allergic disorders. The patch test, however, was extremely helpful in cases of exogenous origin. In conclusion, Dr. Linn said that, although there was not yet a cure for allergic disorders comparable, for example, with the treatment of syphilis with "Salvarsan", or malaria with quinine, they were gradually getting a clearer clinical picture and a better insight into the nature of these diseases, and this, he was confident, was leading them in the right direction towards a more complete cure than was at present available.

DR. H. M. JAY emphasized the importance of recognizing the allergic basis in disorders of the nasal organs and respiratory tract generally, but said that time would not permit any detailed discussion of the subject. He wished to know what Dr. Barlow could say by way of explanation of the noxious effects of *Rhus toxicodendron*. He mentioned the rapidly beneficial effect of chewing leaves of the plant.

DR. P. CHERRY appreciated the usefulness of the paper in regard to the more serious ailments, but would like to hear of something which would help him in dealing with the numerous children suffering from hives.

DR. F. H. BEARE stated that serum rashes after the use of antitetanic and antidiphtheritic and other sera had increased of recent years, and he wished to hear of any explanation.

In reply, Dr. Barlow repeated the statement made in the paper that care and experience were requisite in the interpretation of the weak or indefinite reactions, and that the history often helped in that direction. As regards special methods of preparing vaccines, Dr. Freeman's use of the pathogen-selective method of culturing material was very interesting. Results so far reported did not indicate better results than others were obtaining by the use of special methods of deciding which microorganisms to employ. Dr. Barlow had had good results by obtaining a number of separate strains of streptococci *et cetera* and testing the patient's reaction by intradermal inoculation.

In reply to Dr. Jay's question, he considered that with ivy rash they were dealing mainly with a direct toxic effect and that the chewing of the leaf resulted in absorption of poison with resultant production of antibody. He had not been informed of an increased incidence or severity of serum reactions, and therefore could supply no information at the moment.

In reply to Dr. Cherry, beyond the general management and dietetic supervision mentioned, he did not know of any simple means of dealing with hives in childhood.

A MEETING OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held on August 7, 1935, at the Medical Society Hall, East Melbourne, DR. WALTER SUMMONS, Vice-President, in the chair.

Micturition.

DR. E. GRAEME ROBERTSON read a paper entitled: "A Clinical Study of Micturition" (see page 890). The paper was illustrated with lantern slides and blackboard diagrams.

DR. S. V. SEWELL opened the discussion by congratulating Dr. Robertson on his address and on the success that he and Dr. Denny-Brown had made of the experimental elucidation of such difficult problems. Dr. Sewell had had several short conversations with Dr. Robertson recently and what doubts he had entertained concerning the validity of the deductions were finally dispelled as the result of Dr. Robertson's lucid exposition of the experimental work in his address. The reason for his doubts was that he had got into the habit of looking upon all the viscera and sphincters as being innervated by a first level arc with peripheral ganglion cell mechanism, and by a second level arc through the cord or brain stem, the autonomic outflow through the vagus and pelvic nerves, and by the sympathetic outflow. Knowing that there was no power of inhibiting, for example, the relaxation of the pylorus, it seemed to him to be a big jump to assume that any part of the musculature or sphincters of the bladder could be under the control of the will. Personally, Dr. Sewell had thought that what happened was that control was exerted on the contraction of the *compressor urethræ* muscle through the pudic nerves, and that the only inhibition took place at that level. Dr. Robertson had completely convinced those present that that was not the case. Dr. Sewell had found that in starting micturition it was extremely difficult to exclude any contraction of the abdominal musculature, which contraction had the effect of raising the pressure in the bladder to the extent required to start a rush of proprioceptive impulses to the sacral segments which led to emptying. When the bladder was full, it did seem possible, without abdominal contraction, to start micturition, but only after a very definite interval. Dr. Robertson had suggested that the slowness was due to a delay in contraction and relaxation of involuntary musculature. It was clear that reciprocal action with contraction of the general bladder musculature and relaxation of the internal sphincter was worked at the ganglion peripheral cell level, since stimulation of the pelvic nerves, both in animals and in man, led to the simultaneous contraction of the general bladder musculature and relaxation of the sphincters.

The other point that Dr. Sewell wanted to mention was that he had noticed that when, in spinal injury or disease, a patient's bladder had been allowed to become overdistended and catheterization had not been initiated, it had been difficult to get emptying by automatic contractions, even after the period allowed for spinal shock effects had passed. He would like to know what Dr. Robertson's experience had been in such cases and whether he could throw any new light on the situation. In the reciprocal mechanism of the bladder, when the bladder was in a state of inhibition, the internal sphincter was in contraction, and stimulation of the peripheral end of the cut pelvic nerve brought about contraction of the musculature and relaxation of the sphincter. There was here an example of contraction of the agonist muscle definitely before relaxation of the sphincter. Was this another example of the difference between the reactions of somatic and of visceral muscle?

DR. JOHN T. TAIT said that he had had the advantage of reading Dr. Denny-Brown and Dr. Robertson's papers beforehand, and wished to congratulate Dr. Robertson on his clear exposition of the subject. From the point of view of the practising urologist he felt that most of his difficulties were associated with undue relaxation rather than with retention of urine. The latter usually had an obvious cause, but patients who came to him to be relieved of "unpleasant sensations" were at times very worrying. He could place these patients under two headings. First, there were those with some definite pathological lesion in the bladder or in connexion with the sphincter; and second, those without any definite lesion, but with symptoms attributable to a psychological cause. These two classes of patients required very different treatment. The first called for a warning against over-treatment, either local or over-treatment in the direction of calling too much attention to the workings of the bladder and to their unfortunate condition. He preferred to leave the members of the second group to his friends, the psychologists, but there appeared to be certain conditions of the bladder and rectum in old age in which there might be some regression to the childhood state, with similar disadvantages.

Dr. Tait wished to make two clinical observations and to hear whether Dr. Robertson could explain them. First, in a patient who had a permanent cystotomy opening, the pressure inside the bladder would, of course, be nonexistent; yet these men were liable to recurring attacks of painful spasms with evacuation of urine *per urethram*. He had supposed that these spasms might be due to leakage of urine into the posterior urethra and the setting up of the mechanism of urgent and painful micturition. The second clinical observation was that after prostatectomy one of the most painful procedures of these patients was the onset of similar painful spasms in the bladder, followed by the extrusion of a few drops of urine or of blood while the bladder was widely open. He had noticed that these spasms came on when the rectum was distended with fæces or with flatus, and were relieved by the passage of a rectal tube. Dr. Tait would like to know if Dr. Robertson could offer a neurological explanation of these clinical facts.

DR. H. C. TRUMBLE, after thanking Dr. Robertson for his fine address, presented a number of slides and described some experimental work that he had carried out (part of this had been published in THE MEDICAL JOURNAL OF AUSTRALIA, August 4, 1934, pages 149, 150, and January 26, 1935, pages 118 to 122), which he felt could be reconciled admirably with the views that Dr. Robertson had expressed. Dr. Trumble said that the bladder was developed from the hind-gut, and in reptiles it still retained its connexion with the hind-gut. In the human embryo the connexion was still obvious; in the adult, the bladder and the rectum had separated and opened indepen-

dently to the exterior, but it was to be expected that there would still be certain characteristics common to both. Peristalsis was a function of the gut and continued even when all of the extrinsic nerves were destroyed and degenerated—it was an intrinsic function. It also persisted after cocainization of the gut, but in an irregular, uncontrolled way. The same thing happened after paralysis of the nerves with muscarine or nicotine. It was true that the peristaltic waves might go the wrong way, but it had to be accepted that the waves were not necessarily dependent on nerve participation. Indeed, smooth muscle would still contract after being for a week isolated from the body and in an ice-chest. The bladder musculature also retained the power of contraction when all the extrinsic nerves were destroyed. The contractions resembled, in some ways, peristalsis as seen in the alimentary canal. The usually accepted teaching was that the pelvic nerve activated the detrusor mechanism of the bladder and distal colon, whilst the lumbar sympathetic nerves inhibited these mechanisms. This conception was inadequate to explain certain facts; for instance, he had found that in dogs section of the pelvic nerves resulted in loss of storage of fæces, so that evacuation of small quantities took place at frequent intervals. Retention of fæces did not follow; mass defæcation was lost because storage was deficient and not because the detrusor mechanism was at fault. Langley had shown that the lumbar nerve supply might act as motor or as inhibitor to the colon. The above facts seemed to show that the sacral outflow might contain some inhibitor nerve fibres. Barrington had noted that section of the pelvic nerves in cats was sometimes followed by persistent increase in tone of the bladder musculature. Automatic micturition was similar to the uncontrolled peristaltic emptying of the rectum seen after destruction of the pelvic nerves. Storage of urine, and possibly voluntary restraint of micturition, might be effected through the medium of inhibitor nerve fibres in the pelvic nerves and not in the hypogastric nerves. Dr. Robertson had suggested that control, in the case of the bladder, was of an inhibitory nature and that it was the removal of inhibition that activated the detrusor mechanism.

Dr. Trumble did not attempt to discuss the surgery of the bladder, but briefly described certain attempts he had made to reinnervate experimentally paralysed organs. In dogs, after section of one or both pelvic nerves, he had succeeded in anastomosing the distal ends to the proximal segments of severed hypogastric or obturator nerves. Regeneration of fibres took place and on stimulation the bladder musculature had contracted. So far, nothing of the sort had been attempted in human beings; the difficulties in the way of such an attempt would be very great and the outcome very doubtful.

DR. A. E. COATES, who had been abroad until recently, said that Dr. Robertson's work had been received very well and that it was a fine thing that Dr. Robertson had maintained his critical faculty to such an extent that he had not been afraid to doubt the stated facts and axioms of such famous people as Head and Riddoch. The conclusions that had been reached by Dr. Denny-Brown and Dr. Robertson fitted in with some of the surgical investigations of Learmonth and others. The old idea that there were two sides of the autonomic system with dual dominance had to go by the board. Experimental surgery had upset the importance of the hypogastric rôle; removal of the presacral (hypogastric) nerves was not followed by such serious consequences as it ought to be if these nerves were so important to bladder function. There was some interference with seminal vesicle contraction, however.

Dr. Trumble was one of the great research workers in Victoria and had been engaged for years on important work at the Baker Institute at the Alfred Hospital, and Dr. Coates felt certain that the work would go on until valuable conclusions were reached. Concerning the control by sacral segments in spinal cord lesions, old ideas had been upset, for example, the mass reflex idea. Dr. Coates wondered whether there was in the bladder a combination of voluntary and involuntary control somewhat analogous to that in the face, where the movements of expression were automatic, but voluntary control could be exerted over them.

DR. LEONARD COX also expressed thanks to Dr. Robertson for his stimulating address. Dr. Robertson had mentioned that by volition inhibition was possible. He would like to ask Dr. Robertson if he could explain why the tonic bladder of a highly strung person, which appeared to be able to hold only two or three ounces of urine, could, under the influence of anæsthesia, for example, be made to hold much more.

DR. HAROLD MOORE, in referring to the remark by Dr. Coates on impotence following lumbar ramisection, said that he believed it was due to paralysis or weakness of the internal sphincter. There was some clinical evidence in support of this, for sometimes a patient, after prostatectomy, was able to have intercourse and experience an orgasm, yet there was no emission of semen; then, after the expiration of a variable interval, the patient was able to have normal intercourse with a normal emission. Apparently in the interval the internal sphincter had recovered its tone, as it was obvious that this muscle must be contracted or the semen would simply flow back into the bladder. Dr. Moore asked Dr. Robertson if there was any experimental evidence of any action of the sympathetic on the internal sphincter.

DR. JOHN F. WILLIAMS referred to a statement attributed to an army officer, that under active service conditions one of his biggest trials had been that of having to empty his bladder in public, and to the common experience that many otherwise normal individuals were extremely affected in this function by the presence of other people. Dr. Williams could not see how it could be explained by conscious or unconscious inhibition. There were also many instances of hysterical retention of urine occurring in girls. Another clinical type was the presence of a tonic bladder leading to the constant passing of small quantities of urine. He thought it was Havelock Ellis who had called the bladder "the dynamometer of the emotions". Could Dr. Robertson go any further into these matters?

Dr. Robertson, in reply to Dr. Sewell, said that the voluntary release of micturition was unlike that of contraction of striped muscle: the will was directed to the complete act, and activity of the lower mechanism was necessary before micturition could occur. In spite of the most earnest efforts of will, micturition might in some circumstances be impossible. This bore also upon the problems raised by Dr. Williams. Normal individuals varied greatly in the capacity to inhibit vesical contractions and, when sensation was considered, fluctuations in sensory experience could be explained only as psychological phenomena. At one end of the scale an individual, under experimental conditions, was unable to facilitate vesical contractions—was unable to relax cortical dominance; at the other end of the scale another individual developed high wave-like disturbances which he was not able to inhibit. The acquisition of control during childhood was best explained by the development of inhibition. In regard to the importance of straining in initiating micturition, Dr. Robertson said they were never able to induce micturition by straining in normal subjects, because detrusor activity, with reciprocal relaxation of the sphincter, was not stimulated thereby. He agreed that catheterization was fraught with possibilities of harm, but in one case of their series perfectly developed automatic micturition occurred after a complete transverse lesion of the spinal cord in a sailor who was unable to receive medical attention for three days after the accident; even then it was doubtful whether his bladder had been catheterized. He stated that he had been incontinent of urine after the first few days. In reply to Dr. John Tait, Dr. Robertson said that cases of precipitancy of micturition without urological abnormality of the bladder or sphincters might be present with or without demonstrable neurological lesions. In the former case precipitancy was usually associated with spastic paraplegia, although the tract subserving micturition was probably not the pyramidal tract, though closely associated

with it. Disseminated sclerosis frequently caused such precipitancy, but amyotrophic lateral sclerosis with extreme hypertonicity of extensor muscles was not associated with disorder of urinary function. Experimentally, Barrington had shown that the fibres in the cat were in the most dorsal part of the lateral column, dissociated from the pyramidal fibres. Denny-Brown and Robertson had examined several adults whose only abnormality was precipitancy of micturition or nocturnal enuresis; the patients showed early and powerful development of vesical contractions as a reaction to distension, and they had variable, but diminished, ability to inhibit these contractions; they were also unable to maintain strong contraction of the external sphincter; there appeared to be a defect of cortical control. These might be the subjects of "tonic bladder" to which Dr. Cox had referred. Anæsthesia might increase the vesical capacity in these cases by abolishing reflex activity.

Dr. Robertson said that he was very interested in Dr. Tait's clinical observations, particularly the association of vesical contractions with rectal distension. Experimentally, he and Denny-Brown had shown a close connexion between rectal and vesical activity, particularly after transverse lesions of the spinal cord, but occurring sometimes in the normal subject. Distension of the rectum might excite both rectal and vesical contraction; if the bladder contracted, the internal sphincter and the external sphincter secondarily would relax and so urine might be extruded, particularly if the contraction blocked the suprapubic drainage. The lowering of the sensory threshold or raising of muscular irritability might account for the presence of painful spasms in the absence of rectal contraction.

Dr. Robertson greatly appreciated Dr. Trumble's contribution to the discussion. He felt, however, that a direct muscular effect was not the cause of vesical and rectal contraction. Denny-Brown and he had shown that the bladder or rectal musculature contracted as a whole, and this was associated with exact reciprocation of the sphincter. This surely demanded nervous correlation. In rectal experiments they found peristaltic progress of contractions rare; the rectal muscle contracted synchronously throughout its length. The usual clinical history of abnormal defæcation in *cauda equina* or transverse lesions was constipation, with hard fæces, precipitant and frequent evacuation after purgation. The rectum lacked sufficient power to expel hard fæces, but fluids were expelled as soon as they excited the reaction to distension; it seemed unnecessary to postulate a storage mechanism or actual inhibitory fibres in the peripheral nerves.

In reply to Dr. Coates and Dr. Harold Moore, Dr. Robertson said that the function of the hypogastric nerves was something of a mystery; certainly they conveyed sensation from the bladder, but even that did not appear to be normal desire for micturition. The fact that ejaculation did not occur after section of the hypogastric nerves suggested that they were important in that connexion. Learmonth had shown that only the upper part of the internal sphincter, near the neck of the bladder, contracted when the hypogastric nerves were stimulated; perhaps the absence of this effect, possibly with maintained contraction of the rest of the internal sphincter, allowed semen to flow backwards into the bladder. Some work, as yet unpublished, showed that "funneling of the urethra" was due to progressive dilatation of the internal sphincter from above downwards in a manner directly related to vesical pressure.

Medical Societies.

MELBOURNE PÆDIATRIC SOCIETY.

A MEETING OF THE MELBOURNE PÆDIATRIC SOCIETY was held at the Children's Hospital, Carlton, on August 14, 1935, DR. J. G. WHITAKER, the President, in the chair. The meeting took the form of a series of clinical demonstrations.

Hemihypertrophy.

DR. H. BOYD GRAHAM showed a baby girl, aged twelve months, whose mother had noticed, as soon as she had returned home after confinement, that the baby's right arm and leg were bigger than the left. The baby appeared to be otherwise normal, and it had progressed in a perfectly normal manner till the present time. When the child walked with support, she limped on account of the inequality in the length of the legs. The right lower limb was 18 millimetres (three-quarters of an inch) longer than the left, and the right foot was nine millimetres (three-eighths of an inch) longer than the left. The right arm and side of the face were also proportionately larger than the left side. The disproportion affected the body too. The skin and subcutaneous tissues were quite normal. Radiograms confirmed that there was no dislocation of the hip joints, and also proved that the bones were larger and stronger on the right side. There was no transposition of viscera. There was no family history of similar affection or of twins.

Dr. Graham showed the patient as presenting a rare abnormality, an example of true total congenital hemihypertrophy according to the classification of Stoesser, which he published in the *American Journal of Diseases of Children* in 1928. He said that in true hypertrophy there was variable hypertrophy of soft tissues with enlargement of muscles and increase of underlying bone. The condition might be partial, involving one limb or parts of the body, or total, as in this case. In false hypertrophy the contiguous bones were unaffected and were not lengthened. Dr. Robert Hutchison had reported a case in 1916, in which at autopsy the internal organs on the affected side were also hypertrophied. One theory was that the condition was a mal-development of twin formation. It was difficult to give an accurate prognosis. One observer had eight patients whom he had observed for ten years, and he said that equalization did not occur; but Lockhart Mummery had observed one for nineteen years and there had been progressive equalization. Dr. Graham thought that in odd cases equalization did tend to occur, but in most the hypertrophy remained.

Papilloedema.

DR. MOSTYN L. POWELL demonstrated two cases of papilloedema, each observed after trauma. The first patient was a girl, aged eight years, who had been knocked down by a car in January, 1933. There had been several hours of both pre-accident and post-accident amnesia. The girl had been drowsy and irritable for one week. An X ray picture of the skull revealed no fracture. Two months later the child was admitted to the ward because the mother noticed that the right eye was turning inwards, and the child complained of diplopia. Examination at that time revealed a perfectly happy child with an obvious right rectus paresis. The visual fields were full and the acuity normal, but ophthalmoscopic examination revealed gross papilloedema with a macular tan. The cerebro-spinal fluid was normal. Because of the papilloedema, decompression had been considered, but owing to the well-being of the child it was deferred, the visual acuity being closely watched. The diplopia cleared in hospital. Renal function was investigated because of the retinal exudate, but it was normal. At the present time the child was apparently normal, except for some irritability. Vision was normal. The papilloedema had subsided, but the optic disks were unmistakably pale. There were remnants at the macula of the old exudate. X ray examination of the skull showed no evidence of a calcified hæmatoma.

The second case was that of a boy of twelve years who had been knocked over at school eight weeks previously. He was quite well at the time and had no ill-effects beyond a small hæmatoma on the side of the head. Three weeks later he complained of misty vision, which steadily progressed for a week, when he was admitted to hospital. At this stage his visual acuity was $^6/_{36}$ in both the right and left eyes, and there was papilloedema, 1·5 diopters in the right eye and 1·0 diopter in the left. There was a doubtful contraction of the left upper temporal visual field, but this disappeared later. The results of neuro-

logical examination were otherwise normal, and the boy stated that his vision was now improving. Serological tests gave normal results. The cerebro-spinal fluid was under increased pressure, but was otherwise normal. X ray examination of the skull revealed some doubtful separation of the sutures. Two weeks later, when the vision was improving and the papilloedema was subsiding, the patient developed a clear-cut left lower quadrantic hemianopia and a slight right-sided hemiparesis, with some headache, drowsiness and vomiting. This was the clinical picture at the time of the meeting.

Dr. Powell said that, although the hemianopia and hemiparesis were from opposite sides of the brain, he thought the hemianopia was of more accurate localizing value. The hemiparesis was probably due to pressure of the opposite pyramidal tract against the edge of the *tentorium cerebelli*. Dr. Powell said that the first case was an example of post-traumatic cerebral œdema with papilloedema and secondary optic atrophy. In many head injuries in childhood the condition was thought at the time to be intracranial hæmorrhage, but they subsided quite quickly. Looking back, Dr. Powell thought it wise that operation had not been performed, but he thought that more frequent lumbar punctures should have been done. Over the past year there had been no change in the disk and vision was still perfect, so he thought the prognosis was apparently good.

In the second case Dr. Powell thought there was a cerebral tumour, the symptoms of which were merely precipitated by the injury. The fact that the cerebro-spinal fluid was absolutely normal was, according to C. P. Symonds, against the diagnosis of abscess. Dr. Powell thought that an exploratory operation should be carried out over the posterior aspect of the right temporal lobe.

Sialectasis.

DR. ERIC PRICE showed a girl, aged seven years, who gave a history that for the past five years she had suffered from intermittent swelling of the right parotid gland. The attacks would last from two to seven days, with remissions of one to four weeks. During the attack swelling was always present to some degree, but was worse after a meal, and very painful then. The patient was not ill in the attacks. Examination revealed a moderately firm swelling of the right parotid gland, especially in the upper and anterior part, which was slightly tender. Secretion containing ropy mucus could be squeezed from the duct by gland massage. The orifice was not inflamed. The left side was normal. There were two enlarged hard glands in the right upper deep cervical group. The submaxillary glands were normal. There was no stenosis of the duct and no calculus was seen on X ray examination. A sialogram showed dilatation of some of the finer ductules. Many carious and dead teeth were present in the child's mouth.

A catheter specimen of saliva from the right parotid gland contained pus cells, and on culture streptococci were grown. Dr. Price said he showed the patient because, although the condition was rare, it was well recognized, and Payne, of Saint Bartholomew's Hospital, had reported such cases with sialograms. In this case there was no question of stenosis. Rather was the duct patulous, and this was possibly an ætiological factor. Treatment should be aimed to overcome the two predisposing factors, infection and stasis. The teeth should be attended to and the gland massaged regularly and sialogogues given.

Post-Graduate Work.

THE NEW SOUTH WALES POST-GRADUATE COMMITTEE IN MEDICINE.

Course in Obstetrics and Gynæcology.

THE New South Wales Post-Graduate Committee in Medicine announces that a course of four lectures in obstetrics and gynæcology will be given at the Robert H. Todd Assembly Hall, British Medical Association Building, 135, Macquarie Street, Sydney, commencing on Monday, January 6, 1936.

The lectures will be given by Professor Ludwig Fraenkel, Professor of Gynæcology and Obstetrics, the University of Breslau. Professor Fraenkel has written many papers and is the author of several books on these subjects.

The lectures will begin each evening at 8.15 p.m. The programme is as follows:

Monday, January 6.—"The German Viewpoint of the Treatment of Cancer of the Cervix Uteri."

Wednesday, January 8.—"A Course of Obstetrical and Gynæcological Microscopic Diagnosis." This will include the demonstration of a large number of slides, and those intending to be present are invited to bring any slides they may wish for discussion.

Friday, January 10.—"A General Lecture on Sex Hormones."

Monday, January 13.—"The Practical Application of Hormone Diagnosis and Treatment."

Each lecture will be for one hour, after which questions may be asked and a discussion will be held. The lectures will be illustrated by lantern slides, histological slides and films.

The fee for the course will be £2 2s., or 10s. 6d. for a single lecture. Application to attend the course should be made to the Honorary Secretary, New South Wales Post-Graduate Committee in Medicine, 225, Macquarie Street, Sydney, or payment may be made at the door.

Correspondence.

RADIATION TREATMENT FOR ACNE.

SIR: I have read Dr. Belisario's paper on this subject and also Dr. Molesworth's "note of warning". As a radiologist whose experience dates back to the days when every dermatologist was not his own radiotherapist, I can claim a considerable experience of these cases. I wish to discuss only two points, first the X ray dosage, and secondly the possibility of permanent damage.

As regards the first: my method has been for many years the older one, of administering three one-third doses at weekly intervals, followed by a month's rest and a repetition of the course if necessary. I consider that this method is quite safe, but I am also of the opinion that the method suggested by Dr. Belisario is even safer, for this reason: the radiation effect in any tissue diminishes with increasing rapidity each day after a dose has been delivered. If Dr. Belisario commences with a one-fourth dose, by the end of a week the radiation effect remaining from that dose is very small, and if the one-fourth is repeated at weekly intervals for eight weeks, it can be shown that the total radiation effect at any one time is never anything like a full erythema dose. This diminution in radiation effect Dr. Molesworth appears to leave entirely out of account in his criticism.

Secondly, as to damage. The secreting cells of the sebaceous glands are more sensitive to radiation than the proliferating layers of the skin, and consequently the desired effect on the sebaceous glands can be produced by radiation insufficient to damage the skin. Another example of this difference in sensitivity is seen in epilation, where hair follicles are temporarily damaged without damage to the skin, although they, too, lie deeper than the proliferating layers. Dr. Molesworth speaks of the "normal" sebaceous secretion of these individuals, but sufferers from acne have not a normal secretion, if indeed there is such a thing, but they have in fact a hypersecretion. What one attempts to do is to change the hypersecretion into a more normal one. If Dr. Molesworth would not have any individual's secretion altered

because it was "normal" for that subject, then he would not administer thyreoid for myxœdema, or alkalis for hyperacidity, and he certainly would not use radiation for hyperidrosis or hyperthyreoidism.

Radiation diminishes the activity of the hypersecreting sebaceous glands and consequently cures acne in almost all cases. Taking all the radiological factors into account, I feel that Dr. Belisario's method is as safe, if not safer, than any other I have seen, and in spite of Dr. Molesworth's note of warning, I can conceive of no possible damage which could result to the skin of patients to whom this treatment has been given by a careful operator with properly measured doses.

Yours, etc.,

PHILIP PARKINSON.

British Medical Association House,
135, Macquarie Street,
Sydney,
December 5, 1935.

Proceedings of the Australian Medical Boards.

TASMANIA.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Act*, 1918, of Tasmania, as duly qualified medical practitioners:

Edison, Milton Gray, M.D., 1933 (Univ. Melbourne), Public Hospital, Launceston.

Paterson, Norman Roy, M.B., 1921 (Univ. Sydney), Waratah.

Obituary.

JOSEPH ERNEST GOOD.

WE regret to announce the death of Dr. Joseph Ernest Good, which occurred on December 6, 1935, at Prospect, South Australia.

Medical Appointments.

Dr. P. F. Browne has been appointed Junior Medical Officer, Parkside Mental Hospital, Inspector-General of Hospitals Department, South Australia.

* * *

Dr. W. E. Jones has been appointed, pursuant to the provisions of Section 5 of the *Lunacy Act*, 1928, to be Director of Mental Hygiene, Victoria, for a period of six months from January 1, 1936.

* * *

Dr. F. S. Taylor-Thomas has been appointed District Medical Officer and Public Vaccinator, Mount Magnet, Western Australia.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xviii, xix. and xx.

MOOROOPNA HOSPITAL, MOOROOPNA, VICTORIA: Junior Resident Medical Officer.

MOTHERS' AND BABIES' HEALTH ASSOCIATION, ADELAIDE, SOUTH AUSTRALIA: Medical Officer.

RICHMOND HOSPITALS' BOARD, RICHMOND, NORTH QUEENSLAND: Medical Officer.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associate Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointment and those desiring to accept appointments to any COUNTRY Hospital, are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor", THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such a notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £2 for Australia and £2 5s. abroad *per annum* payable in advance.